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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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CONTENTS

Original Communications

The Use of Plasma for Filling the Pleural Space After Loss of Varying Amounts of Lung. W. E. Adams, M.D., and T. F. Thornton, M.D., Chicago, Ill. ---- 521
On the Management of Burns. Lieutenant Colonel Franklin E. Walton, Medical Corps, U. S. Army ---- 547
Secondary Carcinoma of the Mandible. Captain Raymond E. Buirge, Medical Corps, U. S. Army ---- 553
Jejunostomy for Decompression of the Postoperative Stomach. Arthur W. Allen, M.D., and Gordon Donaldson, M.D., Boston, Mass. ---- 565
Perforation of Postoperative Jejunal Ulcers. K. W. Warren, M.D., and L. S. Fallis, M.D., Detroit, Mich. ---- 569
A New Portable Suction Apparatus for Use With the Miller-Abbott Tube. John A. Gius, M.D., and Clay A. Racely, A. B., Portland, Ore. ---- 574
Multiple Primary Carcinomas of the Gastrointestinal Tract. William I. Sheinfeld, M.D., and Irving Rudolph, M.D., Brooklyn, N. Y. ---- 579
Bilateral Oophorectomy With Radical Operation for Cancer of the Breast. J. Shelton Horsley, M.D., Richmond, Va. ---- 590
Internal Fixation of Fractures of the Patella With Cotton Suture Material. Lyon K. Loomis, M.D., New Orleans, La. ---- 602
Experimental Head Injury Produced by Blasting Caps. Sidney R. Govons, M.D., Philadelphia, Pa. ---- 606
A Case of Infection With Clostridium Sordellii and a Case of Gas Gangrene Treated by Penicillin. H. D. Harvey, M.D., F. L. Meleney, M.D., New York, N. Y. 622
Subparietal Rupture of the Intestine Due to Muscular Effort. Frederick Christopher, M.D., Evanston, Ill. ---- 628
An Accessory Operating Table for Oral Surgery on Infants and Children. Benjamin Goldman, B.S., M.D., F.A.C.S., Erie, Pa. ---- 630
Aseptic Appendectomy—The Application of the Parker-Kerr Suture to the Appendical Stump. Frederic P. Shidler, M.D., San Francisco, Calif. ---- 634
Lesions of the Terminal Colon Associated With Urinary Disturbances. Joseph A. Lazarus, M.D., New York, N. Y. ---- 637
A New Technique for Repair of Facial Paralysis With Tantalum Wire. Willard W. Schuessler, M.D., Washington, D. C. ---- 646
The Association of Thrombophlebitis With Hematogenous Bone Infection. Abraham O. Wilensky, M.D., New York, N. Y. ---- 653
Simple Technique for Multiple Venipunctures. C. Martin Rhode, M.D., Baltimore, Md. ---- 654

Recent Advances in Surgery

The Operative Attack on Organic Peripheral Vascular Disease. I. Ridgeway Trimble, M.D., Wm. S. Cheney, M.D., and Wm. R. Moses, M.D., Baltimore, Md. 655
Book Reviews ---- 679

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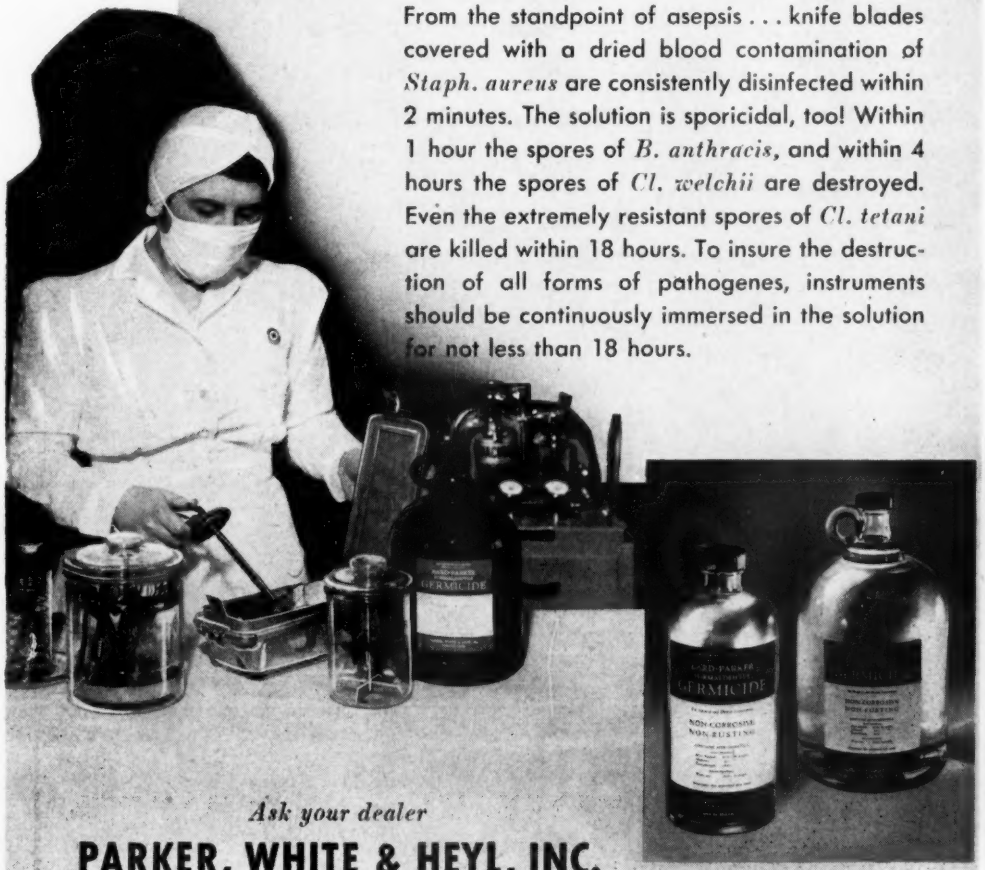

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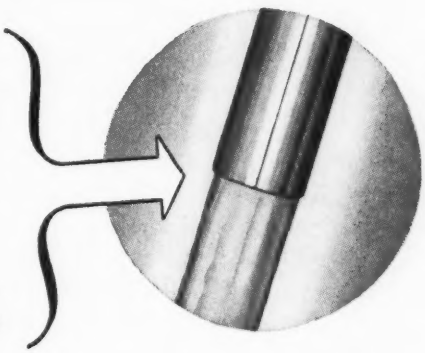

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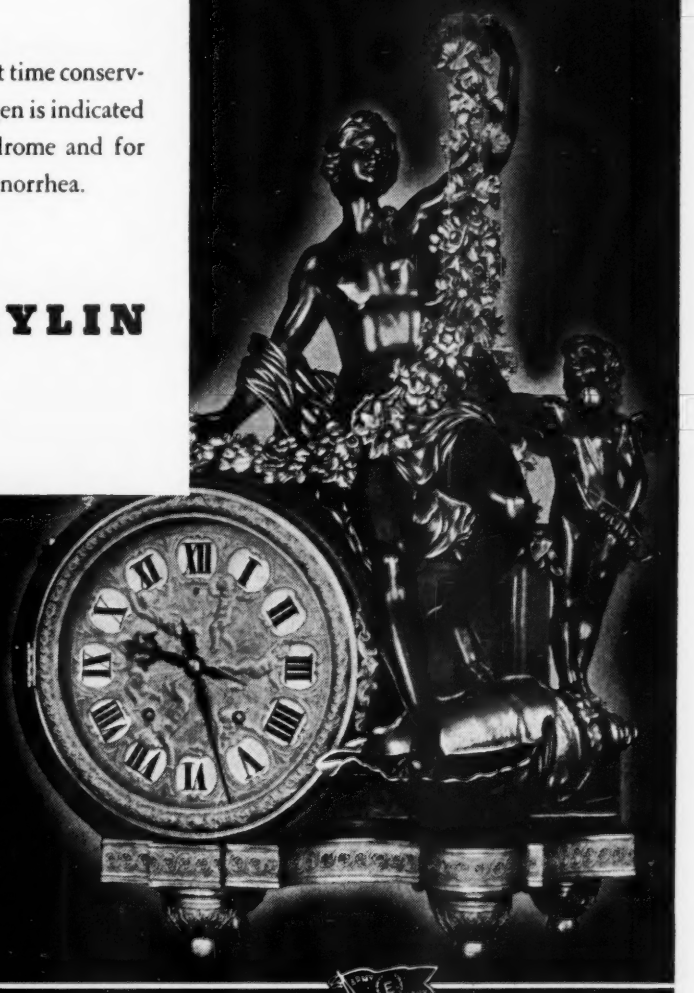

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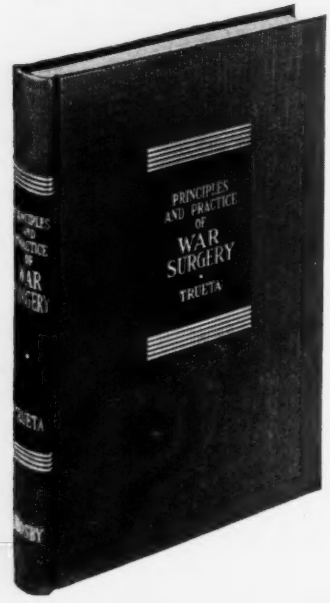

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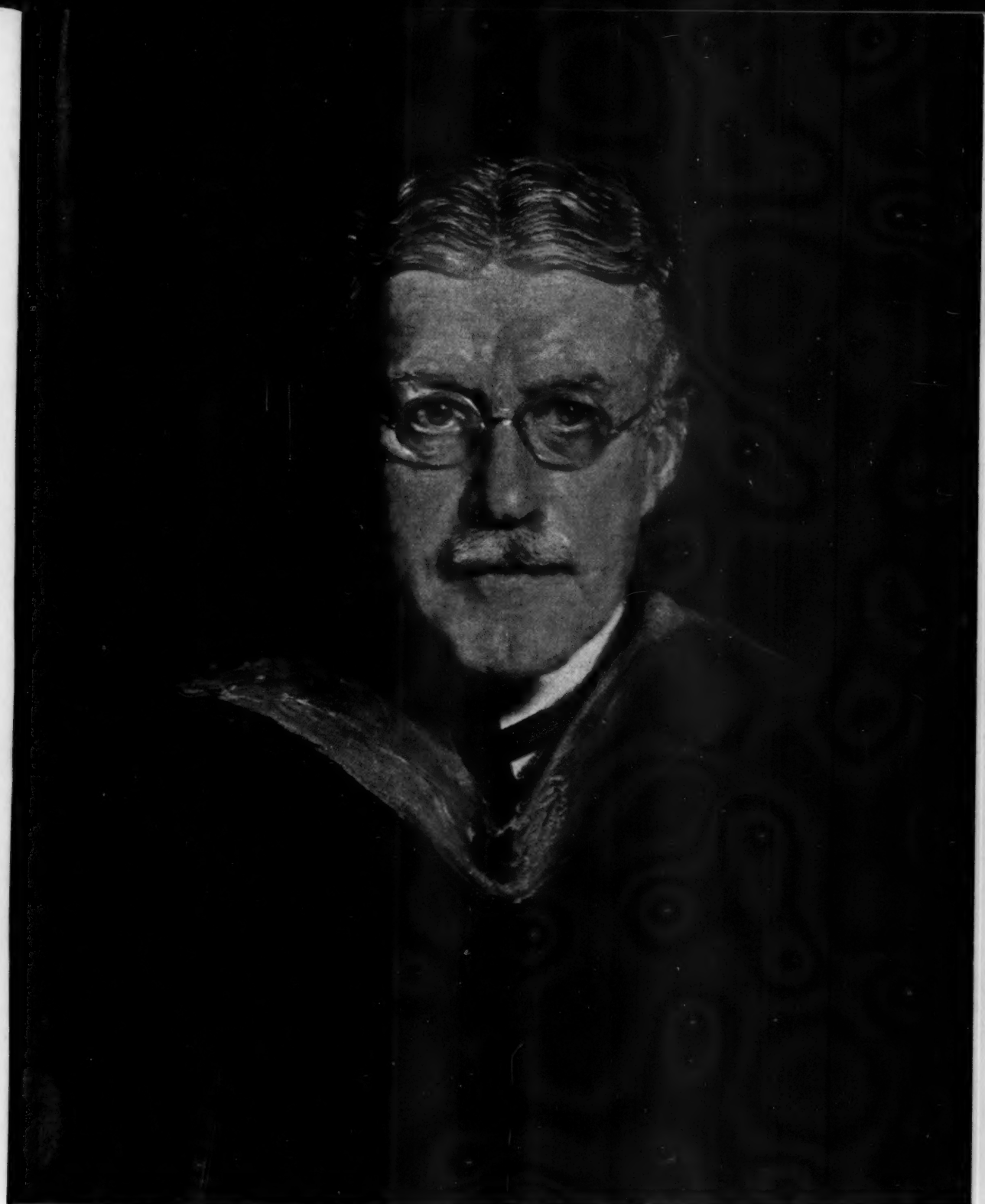

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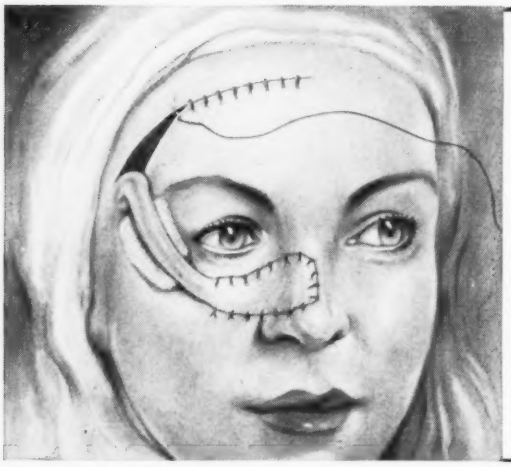

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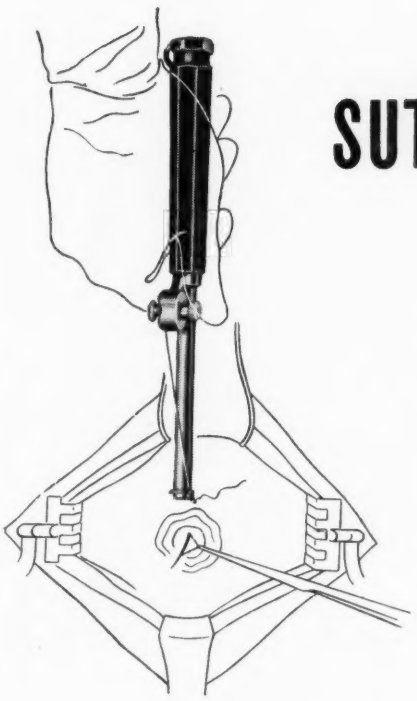

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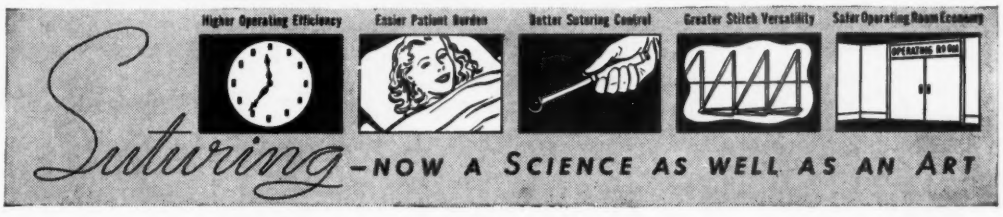
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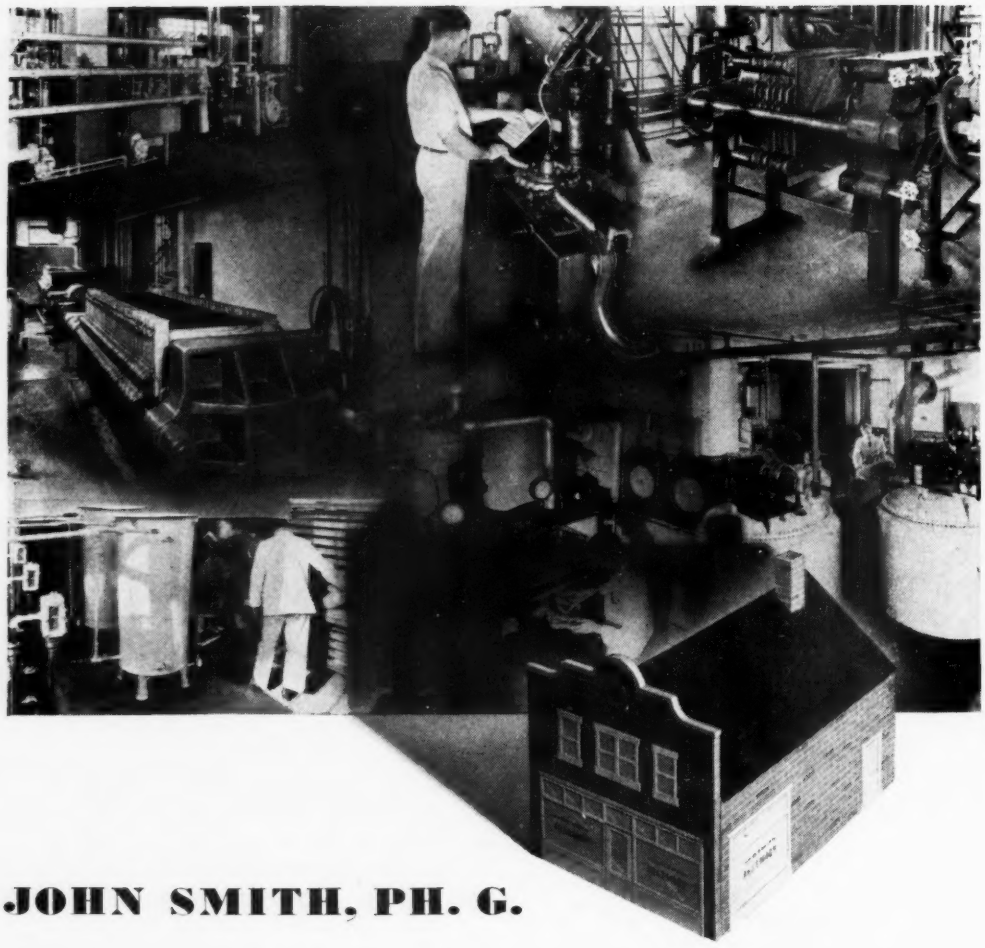

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(From the Department of Surgery of the University of Chicago)

THE surgical treatment of diseases of the thorax has made rapid progress within the past two decades and at the present time operations made through an open pleural cavity are commonplace. Resections of one lobe or of an entire lung for inflammatory conditions and for malignancy are made in increasing numbers. Several factors have contributed materially in the reduction of the mortality of these operations, namely, improvements in diagnostic methods, prevention of shock and severe sepsis, and an understanding of how to maintain the normal function of the organs of the thorax during a surgical pneumothorax.

In spite of the progress made in this field of surgery the present morbidity and mortality rates indicate that further improvement is possible. Better results should be obtained when the various body functions are maintained as nearly normal as possible. In the resection of varying amounts of lung tissue (as with other intrathoracic operations) alteration of the blood may be considerable due to the loss of blood and plasma. Several factors not commonly encountered in operations on other organs may contribute to this alteration, namely, (1) the incision is long and usually divides heavy bellied muscles transversely, (2) vascular adhesions are frequently encountered, and (3) the amount of blood within the lung may be considerable. Following these operations, due to the trauma dealt the chest wall, pleura, etc., both cells and plasma of the circulating blood are lost, part of this being drained away (as much as 600 to 1,200 c.c. following lobectomy) or allowed to accumulate in the pleural space (as in pneumonectomy).

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An analysis of this fluid reveals a slightly lower total protein value than is present in normal blood. Unless replacement therapy is instituted this may lead to a considerable reduction in blood plasma protein, as much as 33⅓ per cent, or to a level as low as 4.0 to 4.5 Gm. per cent. Just how much this might influence the convalescence of the patient would probably vary with the degree of toxicity, the status of the cardiorenal function, etc. (Cardiac failure and cardiorenal failure have been diagnosed as the cause of sudden postoperative exodus when no other cause could be found on post-mortem examination.) It therefore would seem quite logical that the postoperative course of a patient would be less complicated when a more nearly normal blood picture was maintained.

Since resection of the lung is at present frequently indicated (for malignancy, bronchiectasis, lung abscess, and tuberculosis), the factor of blood protein replacement assumes greater importance. In order to determine the method of choice for replacement of protein loss the following plan for experimentation was outlined:

A. Determination of the blood plasma protein fall
   1. Due to anesthesia.
   2. Due to exploratory thoracotomy.
   3. Due to resection of lung tissue with and without the complication of infection.

B. Determination of the replacement therapy needed and the optimum route of administration to prevent the above fall in blood plasma proteins by giving:
   1. Blood transfusions.
   2. Serum transfusions.
   3. Serum intrapleurally.
   4. Serum intrapleurally and blood transfusions.

EXPERIMENTAL METHODS AND PROCEDURES

Animals.—In an attempt to produce hypoproteinemia as seen clinically following lung resections, several different species of animals were tested, namely, rats, cats, rabbits, goats, and dogs. In order to produce a pleural effusion similar to that seen clinically following intrathoracic operations, a number of procedures were employed, such as mechanical and chemical irritation of the pleura, and the leaving of a foreign body (sponge) in the pleural space following a lung resection. Dogs and goats proved to be more adaptable to the problem, and since goats were not readily obtainable in large numbers and were much less economic, most of the experiments were made on dogs. Although an attempt was made to use only healthy animals, pulmonary infection was quite prevalent during the spring and autumn seasons. The presence of infection proved to be of definite value since it offered an opportunity of studying the influence of this factor in the problem under investigation.

Procedure.—Preoperative weight, temperature, and blood studies were routine, the latter including red cells, hemoglobin, hematocrit and total protein determinations. Since the protein loss involved chiefly the albumin factor, after the earlier experiments only a total plasma protein determination was made. In the first few experiments both the Kjeldahl[1] and densimeter falling drop[2] methods for determining plasma proteins were employed. When properly performed these methods checked quite accurately, thus the gravity method was used thereafter. (The values with this method are slightly lower than those obtained by the Kjeldahl because they do not include the nonprotein nitrogen.)

Operation.—After trying several variations of technique, two operative procedures were used, namely, (1) lobectomy using mattress suture technique and (2) pneumonectomy, using the dissection and individual ligation technique in one group and the mattress suture technique in another group. These procedures were similar to those used clinically and produced the desired results. The operative approach was the same for all animals, that is, through the left fifth interspace without rib resection. The muscles were divided in the direction of their fibers thus minimizing the amount of trauma and plasma protein loss in the chest wall wound. Ether anesthesia delivered through an intratracheal catheter under mild positive pressure was routinely employed and was preceded by morphine, 0.015 Gm. subcutaneously. Silk suture material was used throughout. The pleura was massaged with a sponge (2 by 2 inches) which was left in the pleural cavity when the chest wall was closed (see Figs. 1, 2, and 3).

Postoperative Course and Studies.—Following operation the preoperative studies were repeated daily (temperature and plasma proteins) or two to three times a week (hemoglobin, red cells, and hematocrit) for the first two weeks and somewhat less often thereafter. In addition to these studies, thoracenteses and cultures of the pleural fluid were made biweekly along with fluoroscopic and x-ray examinations when necessary (one to three times a week) (see Fig. 4).

The control series of animals included a group of six dogs receiving ether anesthesia (1.25 to 1.5 hours) only, and a second group of eleven dogs having a thoracotomy made, but without lung resection. In other groups a lobectomy was made in seven dogs and a pneumonectomy in sixteen dogs.

The animals receiving replacement therapy were divided into four groups according to the method of management.

In Groups A and B blood or plasma transfusions were given the day of operation and repeated on the first and sometimes the third day following operation. In Group C serum was given intrapleurally at the end of a total pneumonectomy. Group D received the combined treatment of Groups A, B, and C, namely, blood intravenously and serum intrapleurally.

RESULTS OF EXPERIMENTS

A. *Control series.*—In order to evaluate the various factors contributing to the lowering of blood plasma proteins following lung resection, the control experiments were divided into four main groups: (1) anesthesia, 6 dogs; (2) thoracotomy, 11 dogs; (3) lobectomy, 7 dogs; and (4) pneumonectomy, 16 dogs.

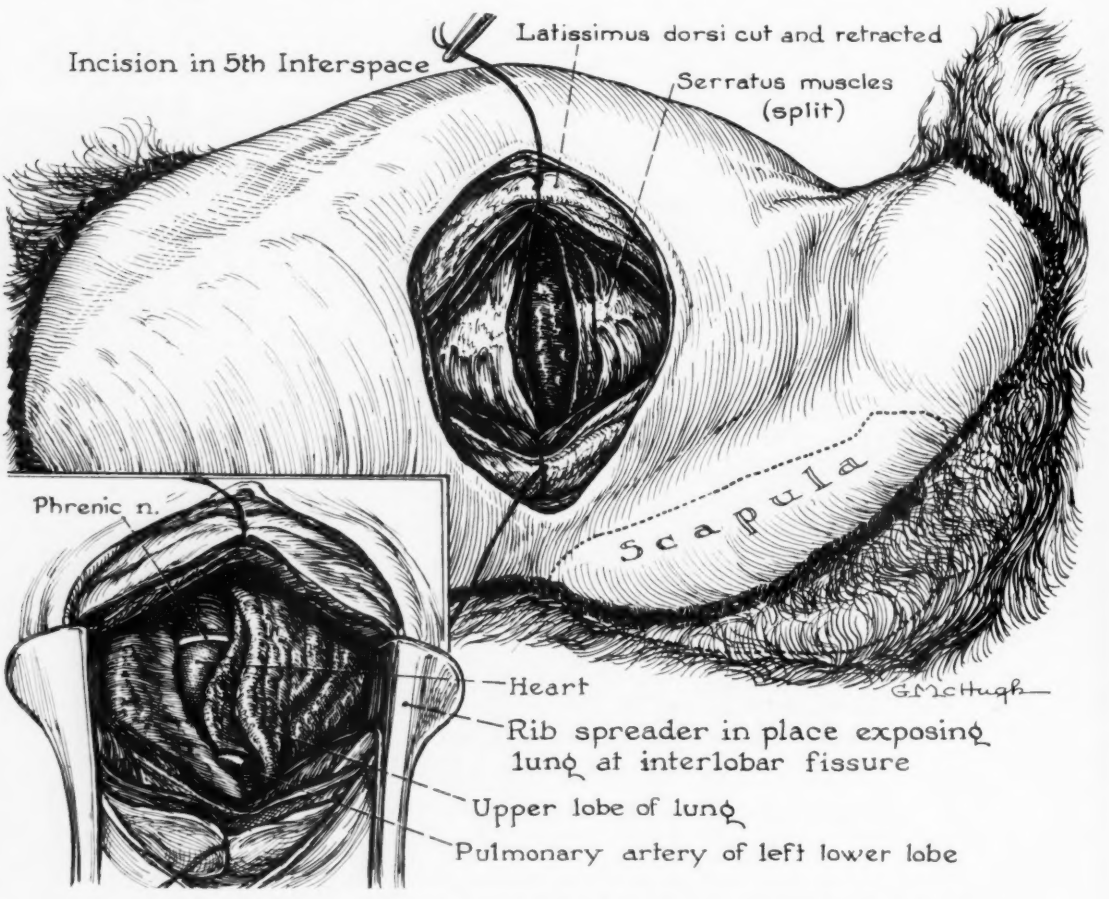


Fig. 1.—Drawing of operative approach for total pneumonectomy through the left fifth interspace. The latissimus dorsi and serratus muscles are divided in the direction of their fibers.

Anesthesia.—Ether anesthesia administered for 1.25 to 1.5 hours produced an average fall in blood plasma proteins of 0.48 Gm. per cent for the six dogs. This usually occurred within the first three days following the anesthesia. There were no fatalities (see Table II).

Thoracotomy.—Thoracotomy wounds without lung resection were made in eleven dogs, a sponge being left in the pleural cavity. In five of these the incision was small as for draining an empyema. The average fall in blood plasma proteins for this group was 0.45 Gm. per cent (0.35, 0.74, 0.66, 0.10, and 0.43 Gm. per cent). In six other dogs the incision

gave wide exposure such as needed for lung resection. The average fall in this group was 0.85 Gm. per cent (0.93, 0.91, 0.91, 0.70, 0.87, and 0.80 Gm. per cent). The lowest blood plasma protein reading usually occurred on the second to the fourth postoperative day. A late chest wall infection caused the only death in the group of 11 dogs (see Table II).

TABLE I

SUMMARY OF PLASMA PROTEIN STUDIES FOLLOWING LOBECTOMY AND PNEUMONECTOMY IN DOGS

	NO. DOGS	INFECTED	INFECTED GROUP PLASMA PROTEIN FALL		NONINFECTED GROUP PLASMA PROTEIN FALL	
			AVERAGE	RANGE	AVERAGE	RANGE
A. Reduction of plasma proteins in lobectomy and pneumonectomy						
1. Ether anesthesia alone	6	0	–	–	0.48	< 0 – 0.97
2. Exploratory thoracotomy						
small	5	0	–	–	0.45	< 0.10 – 0.74
large	6	2	0.93 0.91		0.82	< 0.70 – 0.92
3. Lobectomy mattress suture	7	5	1.50	< 1.01 – 2.47	0.28 0.38	
4. Pneumonectomy—						
mattress suture	10	5	1.20	< 0.28 – 1.88	1.34	< 0.42 – 1.99
individual ligation	6	2	1.36 2.05		1.30	< 0.80 – 1.60
B. Replacement of plasma protein loss in lobectomy and pneumonectomy						
1. Lobectomy (mattress suture)						
(a) Serum transfusion	8	5	0.58	< 0 – 1.40	0.28	0 – 0.74
(b) Blood transfusion	4	3	0.63	< 0 – 1.33	0.28	
2. Pneumonectomy (mattress suture)						
(a) Serum transfusion	5	3	1.05	< 0.74 – 1.50	0.43 2.33	
(b) Blood transfusion	2	1	1.29		0.84	
3. Pneumonectomy (individual ligation)						
(a) Serum transfusion	6	1	0.77		0.56	< 0.15 – 1.53
(b) Serum intrapleurally						
200 c.c.	11	3	1.0	< 0.74 – 1.22	0.79	< 0.24 – 1.29
400 c.c.	13	7	0.62	< 0 – 1.26	0.52	< 0 – 1.58
(c) Blood transfusion (100 c.c.) and serum intrapleurally (200 c.c.)	8	3	0.34	< 0 – 0.53	0.44	< 0 – 1.11

Lobectomy.—The operation of lobectomy was made, using two different techniques, but it was soon found that the mattress-suture technique was the only one applicable for the experiment. In seven dogs, the left

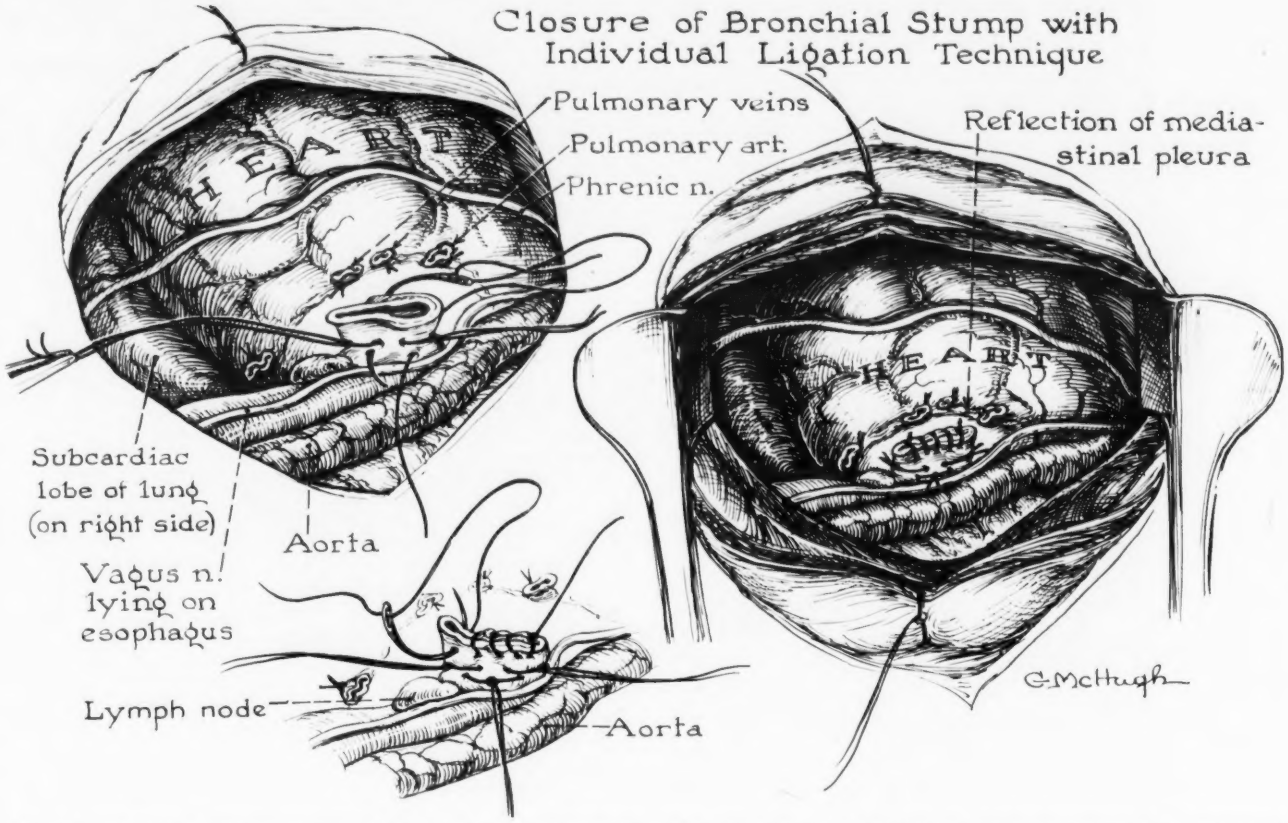


Fig. 2.—Drawing showing closure of the left primary bronchus with two rows of sutures in pneumonectomy by individual ligation technique.

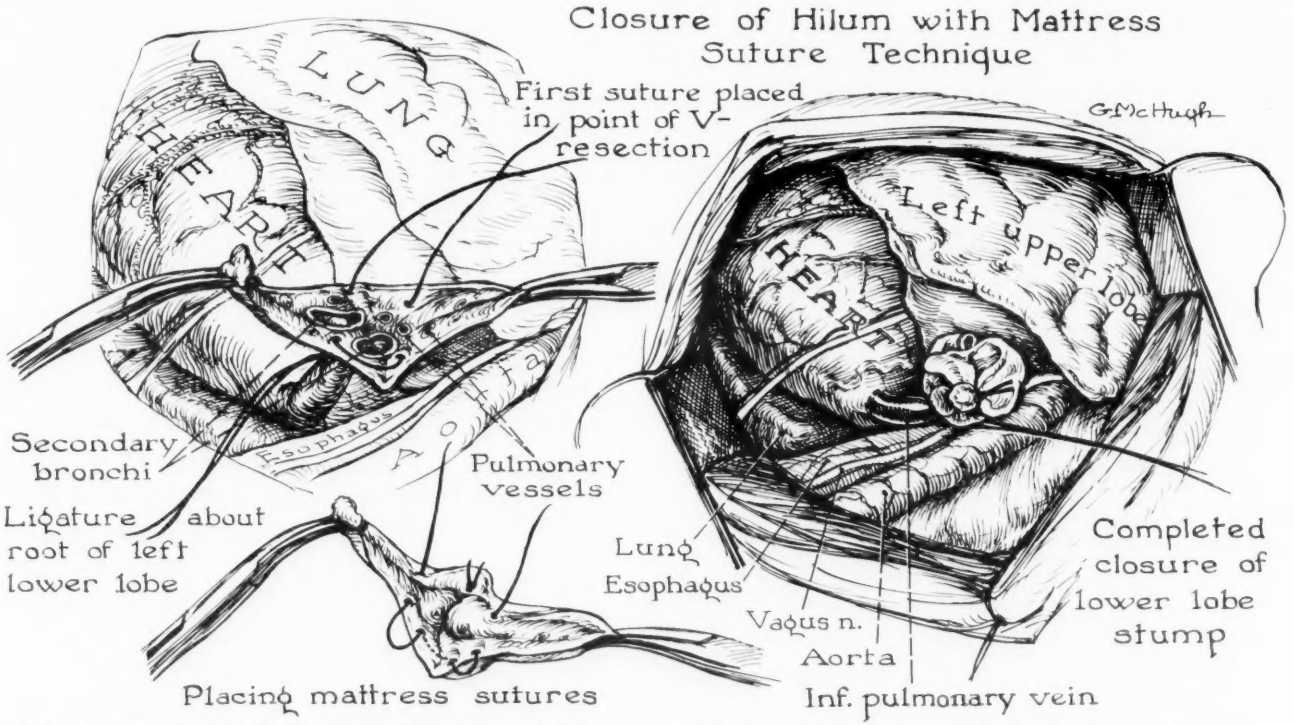
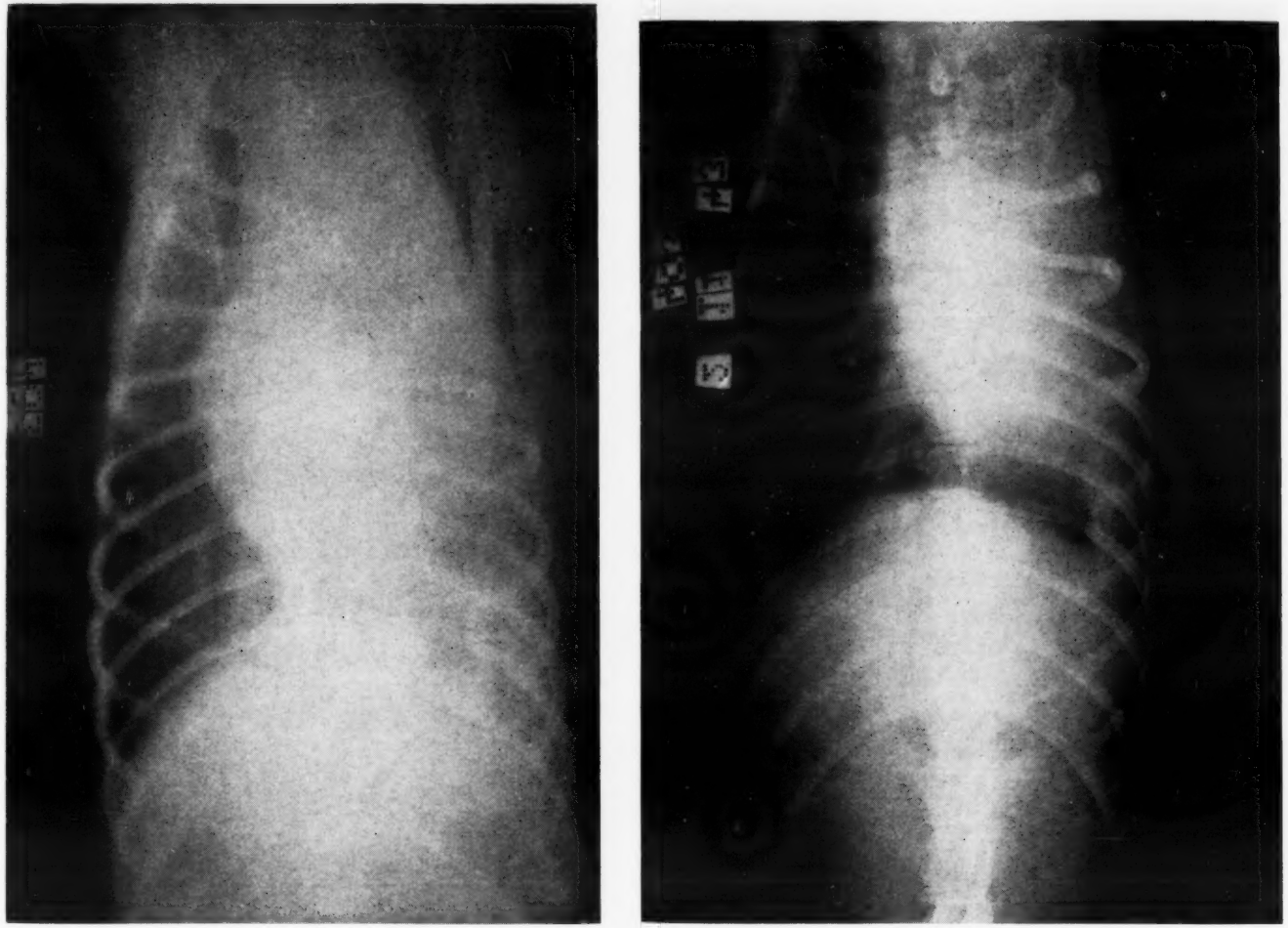


Fig. 3.—Drawing to illustrate mattress-suture technique of closing the bronchi and vessels in left lower lobectomy.

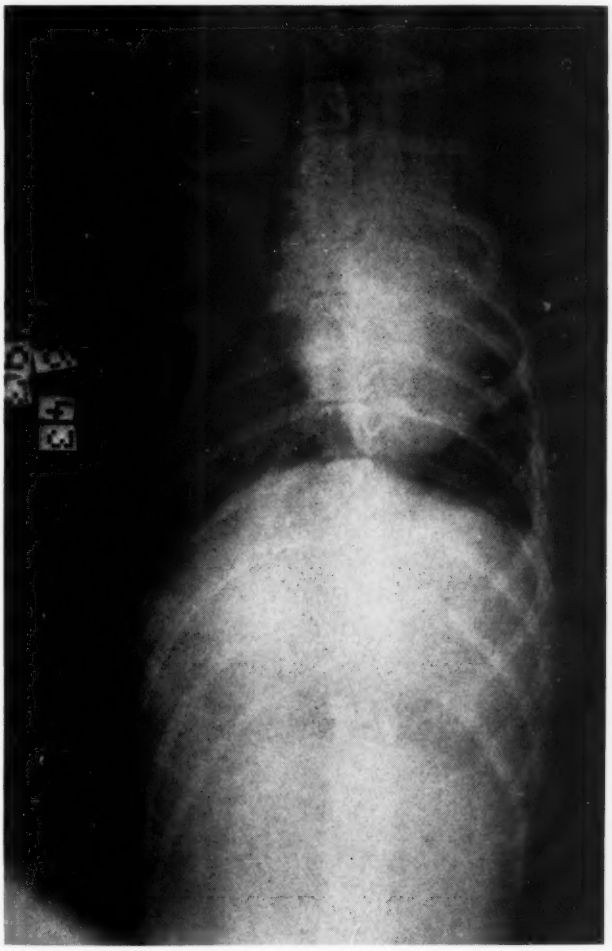


A. *B.*

Fig. 4 *A*, *B*, and *C*.—X-rays of the chest of Dog 493 taken one day, seventeen days, and forty-two days respectively following total pneumonectomy (left) by the individual ligation (dissection) technique.

Note opacity of left pleural space on first day following operation (200 c.c. of serum were placed in this space at the conclusion of the operation). The subcardiac lobe was more evident in the second x-ray and the opaque area was markedly diminished in size. The third x-ray revealed further diminution of left pleural opacity with deviation of the heart toward the left thoracic wall and further expansion of the subcardiac (accessory) lung lobe. On sacrificing the animal the following day, only 5 c.c. of clear serum remained in the left pleural space. There was no pneumothorax.

lower lobe was removed, five of which were infected. The infection was usually on the basis of distemper or empyema following operation. The average fall in blood plasma protein for this group was 1.5 Gm. per cent, the range of fall varying from 1.01 to 2.47 Gm. per cent. The lowest plasma protein level for the group was 3.96 Gm. per cent and was observed on the second day following operation. (See Table III, Dog 270.)



C.

Fig. 4*C.*—*(For legend see opposite page.)*

Pneumonectomy.—The operation of pneumonectomy was performed using two different techniques for closing the bronchial stump, namely, mattress suture, and individual ligation techniques. In the first group of ten dogs where the bronchial stumps were closed by multiple mattress sutures, five were complicated by an infection due to distemper or empyema. The average fall in blood plasma proteins for this group was 1.34 Gm. per cent of the noninfected, and 1.20 for the infected group. The lowest plasma protein level was 3.96 Gm. per cent (see Table III,

TABLE II

REDUCTION OF TOTAL PLASMA PROTEINS BY ANESTHESIA AND THORACOTOMY

DOG NO.	INITIAL WEIGHT (KG.)	INITIAL TOTAL SERUM PROTEIN	INITIAL HEMATOLOGIC FINDINGS			INFECTION PRESENT	PLEURAL FLUID REMOVED (C.C.)	LOWEST TOTAL SERUM PROTEIN (GM. %)	TIME FOLLOWING PROCEDURE (DAYS)	AMOUNT OF FALL IN P.P. (GM. %)	HEMATOLOGIC FINDINGS AT TIME OF LOWEST PLASMA PROTEIN LEVEL			DIED OR SACRIFICED	REMARKS
			HEMATOCRIT	HB.	R.B.C. M.						HEMATOCRIT	HB.	R.B.C. M.		
Ether anesthesia for 1.25 hours in 6 dogs															
349	17.7	5.14	40	14.5	5.6	–	–	5.35	2	0	41	14.0	6.4	S	
350	16.3	5.39	41	14.5	7.0	–		5.28	1	0.11	42	14.5	6.6	S	
316	6.8	5.25	58	14.	8.9	–		4.73	2	0.52	51	12.5	7.8	S	
338	9.0	6.85	43	–	7.8	–		5.88	2	0.97	43	–	8.0	S	
384	8.5	6.30	46	–	8.6	–		5.60	3	0.70	36	–	7.0	S	
385	17.2	6.33	62	16	9.0	–		5.74	3	0.59	58	15.0	7.8	S	
Average fall in plasma proteins—0.48 Gm. %															
Exploratory thoracotomy—small incision in 5 dogs															
379	8.2	5.35	–	–	–	–	0	5.00	1	0.35	–	–	–	S	
382	7.5	5.64	–	–	–	–	300	4.90	2	0.74	–	–	–	S	Sponge left in chest; temperature elevation of 1 to 2° F.
383	7.8	5.46	–	–	–	–	400	4.80	3	0.66	–	–	–	S	
385	17.6	5.80	–	–	–	–	200	5.70	2	0.10	–	–	–	S	
388	13.1	5.60	–	–	–	–	0	5.18	3	0.42	–	–	–	S	
Average fall in plasma proteins—0.45 Gm. %															
Exploratory thoracotomy—large incision in 6 dogs															
378	13.9	5.31	43	12.5	7.6	+	0	4.38	4	0.93	40	10.0	7.0	D	
379	6.9	5.88	47	10.5	7.8	±	0	4.97	4	0.91	49	11.0	8.0	S	No sponge left in chest, chest clear on fluoroscopy; temperature elevation of 1 to 2° F.
382	7.8	5.39	48	10.5	7.6	–	0	4.48	3	0.91	48	10.5	8.0	S	
383	8.25	5.74	46	12.0	7.6	–	0	5.04	2	0.70	44	10.0	7.2	S	
388	12.7	6.01	45	16.0	7.2	–	0	5.14	4	0.87	43	15.0	6.7	S	
389	8.5	5.56	42	15.5	6.2	–	0	4.76	3	0.80	41	15.0	6.4	S	
Average fall in plasma proteins—0.85 Gm. %															

Dog 430). In the second group of six dogs, where the stump was closed by individual ligation technique, only two dogs were infected, one due to distemper, the other due to an open bronchus. The average fall in blood plasma protein for this group was 1.31 Gm. per cent for the non-infected and 1.70 Gm. per cent for the infected. The greatest fall usually appeared between the second and fourth days following operation. The range of fall in plasma proteins was from 0.8 to 2.05 Gm. per cent. The lowest plasma protein level observed was 4.24 Gm. per cent (see Table III, Dog 389). A large percentage of the animals in the control series showed a fall in plasma proteins to a level between 4.0 and 4.75 Gm. per cent.

Thus, in summarizing the results of the control series, it is seen that the lowering of blood plasma protein following the resection of lung tissue is in part due to (1) the anesthesia, (2) the operation on the chest wall, (3) removal of lung tissue per se, and (4) infection when present, the amount of fall being almost the same following pneumonectomy as following lobectomy.

B. *Replacement.*—These experiments fell into three main groups: namely: (1) lobectomy, (2) pneumonectomy with mattress suture technique, and (3) pneumonectomy with individual ligation technique.

Lobectomy.—Twelve animals were included in this group of lobectomies, eight of which received serum intravenously, and four received whole blood transfusions as their replacement therapy. This was usually administered in 200 c.c. amounts of serum, and approximately 165 c.c. of whole blood intravenously at any one time. The replacement material was usually given on the day of operation and on the second and third days following operation. Of the eight animals receiving serum, five were infected, three due to distemper, one due to chest wall infection, and the fifth to empyema. The average fall in blood plasma proteins for these five animals was 0.58 Gm. per cent, the range being from 0 to 1.40 Gm. per cent. The three noninfected dogs averaged a fall of 0.28 Gm. per cent with a range of 0 to 0.70 Gm. per cent.

Of the four animals receiving whole blood, three were infected, two due to bronchial stump leak and one to distemper. The average fall in blood plasma protein for these three animals was 0.63 Gm. per cent, the range being from 0 to 1.33. The noninfected animal had a fall of only 0.28 Gm. per cent.

Pneumonectomy—Mattress-suture Technique.—In this group of seven dogs, in which mattress-suture technique was used, five received plasma intravenously, and two, whole blood intravenously. Of the first five, three were infected, two of which were due to distemper, and one due to empyema. The average fall for the three infected was 1.05 Gm. per cent. The fall for the two non-infected was 1.2 Gm. per cent. Of the two receiving whole blood intravenously, one was infected (empyema). The blood plasma protein fall in this case was 1.29 Gm. per cent. The noninfected animal had a fall of 0.84 Gm. per cent.

TABLE III

REDUCTION IN TOTAL PLASMA PROTEINS BY LOBECTOMY AND PNEUMONECTOMY

DOG NO.	INITIAL WEIGHT (KG.)	INITIAL TOTAL SERUM PROTEIN	INITIAL HEMATOLOGIC FINDINGS			INFECTION PRESENT	PLEURAL FLUID REMOVED (C.C.)	LOWEST TOTAL SERUM PROTEIN (GM. %)	TIME FOLLOWING PROCEDURE (NO. DAYS)	AMOUNT OF FALL IN P. P. (GM. %)	HEMATOLOGIC FINDINGS AT TIME OF LOWEST PLASMA PROTEIN LEVEL			DIED OR SACRIFICED	REMARKS
			HEMATOCRIT	HB.	R.B.C. M.						HEMATOCRIT	HB.	R.B.C. M.		
Left lower lobectomy (mattress suture) in 7 dogs															
346	10.5	5.74	49	16.5	7.3	–	0	5.46	7	0.28	43	15.0	7.3	S	
262	11.7	5.46	53	15.5	7.5	–	120	5.14	4	0.32	48	17.0	6.5	S	
338	10.5	5.60	36	13.5	5.1	+	110	4.31	3	1.29	36	14.5	6.1	S	Small empyema healed
347	11.6	5.70	51	17.0	7.2	+	212	4.69	3	1.01	49	15.0	8.3	D	Empyema
221	12.0	6.05	–	–	–	+	400	4.52	3	1.53	46	13.4	5.8	D	Empyema
315	18.5	6.85	–	–	–	+	500	4.38	3	2.47	45	13.5	5.9	S	Empyema, healed by aspiration
270	13.0	5.18	47	16.0	5.8	+	410	3.96	2	1.22	40	13.5	5.5	S	Wound infection
Average fall—1.16 for 7 dogs															
Left total pneumonectomy (mattress suture) in 10 dogs															
323	20.0	6.16	46	16.0	5.8	+	855	4.76	3	1.40	38	15.5	5.1	D	Empyema
324	14.2	5.98	46	16.5	7.5	+	780	4.10	6	1.88	31	12.0	5.4	D	Distemper, chest wall infection
329	11.3	6.22	40	16.0	7.6	+	150	4.86	2	1.26	38	15.5	5.6	D	Distemper
330	9.2	5.35	34	13.5	5.1	+	285	5.07	2	0.28	38	14.5	5.4	D	Distemper empyema
429	7.7	6.12	51	15.0	7.1	+	400	4.94	1	1.18	–	–	–	D	Distemper empyema
208	14.7	6.12	45	14.5	8.5	–	60	5.70	3	0.42	48	16.0	7.8	S	

343	20.2	5.98	47	15.5	8.3	–	110	4.24	2	1.74	33	12.5	5.0	D	**Died of tension pneumothorax**
327	15.1	5.80	49	15.5	6.2	–	480	4.76	3	1.04	49	18.0	7.9	D	Died of tension pneumothorax
328	15.2	6.26	42	13.5	6.3	–	975	4.73	6	1.53	39	14.0	6.3	D	Died of tension pneumothorax
430	16.1	5.95	46	14.5	6.8	–	?	3.96	1	1.99	–	–	–	D	Hemorrhage into pleural space
Average fall—1.27 for 10 dogs															
Left total pneumonectomy (individual ligation) in 6 dogs															
394	12.0	6.40	58	14.5	6.0	–	188	4.80	3	1.60	54	14.0	5.8	S	
389	8.5	5.53	58	16.5	7.4	–	370	4.24	3	1.29	60	14.0	6.2	S	
424	10.1	5.28	56	12.5	6.0	–	135	4.48	2	0.80	52	13.5	5.8	S	
423	8.1	6.30	63	14.0	7.0	–	210	4.86	7	1.44	63	15.5	7.5	D	Died of hemorrhage from femoral artery after drawing blood
562	10.8	6.47	62	16.0	6.4	+	200	5.11	2	1.36	–	–	–	D	Died of distemper on 7th P.O. day
293	13.7	6.64	40	12.0	5.2	+	90	4.59	4	2.05	33	11.0	4.0	D	Open stump and empyema
Average fall (plasma proteins)—1.42 Gm. % in 6 dogs															

TABLE IV

REPLACEMENT OF PLASMA PROTEIN LOSS IN LOBECTOMY

DOG NO.	INITIAL WEIGHT (KG.)	INITIAL TOTAL SERUM PROTEIN	INITIAL HEMATOLOGIC FINDINGS			REPLACEMENT THERAPY (C.C.)	INFECTION PRESENT	PLEURAL FLUID REMOVED (C.C.)	LOWEST TOTAL SERUM PROTEIN (GM. %)	TIME FOLLOWING PROCEDURE (DAYS)	AMOUNT OF FALL IN P.P. (GM. %)	HEMATOLOGIC FINDINGS AT TIME OF LOWEST PLASMA PROTEIN LEVEL			DIED OR SACRIFICED	REMARKS
			HEMATOCRIT	HB.	R.B.C. M.							HEMATOCRIT	HB.	R.B.C. M.		
Left lower lobectomy with serum transfusion in 8 dogs (mattress sutures)																
440	12.8	5.67	58	15.0	6.8	600	–	490	5.60	4	0.07	50	13.0	5.6	S	
454	7.0	4.55	48	16.0	6.2	400	–	0	4.76 5.18	9 3	0	43	14.0	5.7	S	
479	7.8	6.09	70	16.5	8.2	400	–	120	5.35	3	0.74	59	15.0	7.3	D	Died of bilateral pleural effusion
459	7.0	5.21	57	14.0	5.9	400	–	170	4.94	1	0.27	54	14.0	5.7	D	Distemper third P.O. day
441	10.4	5.53	55	16.0	7.1	600	+	435	5.18	4	0.35	49	14.0	6.1	D	Died of distemper 5 days P.O.
453	7.5	5.56	48	15.5	6.1	600	+	80	4.16	9	1.40	43	14.5	5.8	D	Died of distemper 12 days P.O. No fall in proteins until ninth day
458	8.6	5.28	65	16.0	7.3	400	+	995	4.38	4	0.90	56	15.0	6.6	D	Died of chest wall infection
480	12.0	5.95	68	16.0	7.4	400	+	810	6.16 6.01	3 2	0	56	15.0	6.3	D	Died of empyema
Average fall in plasma proteins (except for Dog 453)—0.33 Gm. % in 7 dogs																
Left lower lobectomy with blood transfusion in 4 dogs (mattress suture)																
490	6.5	4.80	57	16.0	6.4	330	–	60	4.52	1	0.28	64	17.0	7.3	S	
486	7.4	5.28	56	16.0	6.1	330	+	205	5.31 5.60	2 1	0	--	--	--	D	Distemper 3 days P.O.
487	7.3	6.16	53	15.5	6.2	330	+	290	4.83	1	1.33	--	--	--	D	Bronchial leak
491	10.7	5.95	54	15.5	6.1	330	+	522	5.39	2	0.56	62	17.0	7.0	D	Empyema
Average fall in plasma proteins—0.54 Gm. % in 4 dogs																

The seven animals having pneumonectomy received only one-half as much replacement therapy as the preceding lobectomy group, thus explaining the greater fall in blood protein. In spite of this, a plasma protein level of approximately 5.0 Gm. per cent or above was maintained in all of the dogs (except the two receiving whole blood replacement).

Pneumonectomy—Individual Ligation Technique.—This group of thirty-eight animals in which individual ligation technique was used was divided into three parts.

1. Part one included six animals which received 400 c.c. of serum intravenously in two injections. Only one of this group was infected, this being due to an open bronchus. Of the five noninfected dogs, the average fall in plasma proteins was 0.56 Gm. per cent, the range being from 0.15 to 1.53. The one infected animal had a fall of 0.77 Gm. per cent. The lowest level for this group was 4.83 Gm. per cent.

2. In part two, twenty-four animals were subdivided into two groups.

Group A. Eleven dogs received 200 c.c. of serum intrapleurally at the time of operation. Three of these were infected; one had distemper and the other two developed an empyema. Of the three infected, the average fall was 1.0 Gm. per cent. For the remaining eight noninfected dogs, the average fall was 0.79 Gm. per cent, the range being from 0.24 to 1.29.

In Group B each of thirteen dogs received 400 c.c. of serum intrapleurally at the time of operation. Seven of these thirteen were infected; five had distemper and two developed an empyema. The average fall for the infected group was 0.62 Gm. per cent, the range being from 0 to 1.26. For the non-infected group of six dogs, the average fall was 0.52 Gm. per cent, with a range from 0 to 1.58 Gm. per cent. Only one of the thirteen animals receiving 400 c.c. of serum intrapleurally had a blood plasma protein level below 4.85 Gm. per cent and only four below 5.0 Gm. per cent.

3. In part three, eight animals each received 200 c.c. of serum intrapleurally at the time of operation and 100 c.c. of blood intravenously. Of the eight animals, three were infected; one had distemper, the other two developed an empyema. The average fall for these three was 0.34 Gm. per cent, with a range of 0 to 0.53. The average fall for the five noninfected was 0.44 Gm. per cent, with a range of 0 to 1.11. The reason for the infected group not falling more was probably that most of the animals had a very low plasma protein level before operation; namely, 4.06, 4.85, and 5.64.

In summarizing the results for the treated series, several facts were brought out: A marked fall in blood plasma proteins could usually be prevented by the intravenous administration of either serum or whole blood in sufficient amounts. A similar result could be obtained by the use of adequate amounts of serum intrapleurally. In infected animals a greater fall was observed than in the noninfected and could not be

entirely prevented by replacement therapy. The level of blood plasma proteins was usually maintained above 5.0 Gm. per cent by the replacement therapy. In infected animals it was occasionally below this level. In some animals the postoperative plasma protein level was well above that found before operation (see Table V, Dogs 540, 541, 575, and 597).

DISCUSSION

A review of the results of the experiments indicates that several contributory factors were involved in the production of hypoproteinemia following the resection of lung tissue. Exploration of the pleural cavity through an incision in the chest wall produced a definite lowering of the blood plasma proteins, on an average of approximately 0.75 Gm. per cent. This amount of reduction was greater when less attention was paid to the amount of trauma to the chest wall. The size of the incision apparently had a definite influence on how much lowering one could expect.

Preliminary to the exploration of the chest, ether anesthesia appeared to produce a definite lowering in blood plasma proteins, usually averaging between 0.25 and 0.5 Gm. per cent. A third factor which played a definite role was infection. As a rule, this caused a drop of as much as 25 per cent more than was noted in the clean cases. In some animals where distemper was already present, this fall in plasma protein level occurred before operation. Complications such as empyema, leak of the bronchial stump, and chest wall infection were more prone to occur where distemper was present. The fact that the blood protein level was quite low before operation appeared occasionally to prevent marked lowering by the operative procedure. This explains some of the discrepancies in fall of plasma proteins following a similar procedure in different animals. The size of the animal, per se, appeared to have relatively little or no influence. The blood picture was usually altered in the same direction as the plasma proteins, that is, a fall in red cell count as well as hemoglobin and hematocrit figures.

Hypoproteinemia almost always occurred within twenty-four to forty-eight hours following operation and usually persisted seven to ten days. In some of the experiments, death occurred from complications, such as distemper, bronchial stump leak, or empyema, too early to show the full effect of the operation on the blood protein level.

Replacement Therapy.—Early in the course of the experiment it was found that one could almost routinely prevent a marked fall in plasma proteins by the use of adequate amounts of either serum or whole blood given intravenously. Likewise, serum given intrapleurally in sufficient amounts prevented a marked fall in plasma proteins following the resection of lung tissue in the majority of cases, although not as efficiently as when blood or serum were given intravenously. The total amount of whole blood used to prevent hypoproteinemia was approximately 10 to

15 c.c. per kilogram of body weight. When serum was administered intrapleurally, larger amounts were necessary to obtain similar results.

Only four goats were used in the experiment, two being controls and two receiving replacement therapy. Little difficulty was experienced regarding the operative procedure per se and no infection of any magnitude was encountered in this animal. The two control animals showed a similar trend to that observed in the dog; namely, a fall in plasma proteins following lobectomy and pneumonectomy, of 1.53 and 1.77 Gm. per cent respectively.

Replacement therapy used in the other two goats was serum intravenously, 450 c.c. in one animal, and 400 c.c. intrapleurally in the second animal. In the first of these there was only 0.75 Gm. per cent fall in plasma proteins. In the second, due to a small encapsulated empyema, the fall was greater, although less than would have occurred in a goat with a similar operation, but without replacement therapy. The fall of proteins in this animal was also probably partially due to the lack of sufficient serum given by the intrapleural route. The plasma protein level maintained following operation was 5.64 and 5.21 Gm. per cent respectively. In both dogs and goats when replacement therapy was given, lowering of blood protein when present was very transient, usually lasting only one or two days as compared to eight or ten in the control series.

Clinical studies of blood plasma proteins before and following operations on the chest, including the resection of a part of the lung and as yet unpublished, reveal results similar to those described previously in dogs and goats. One patient, a 9-year-old boy, showed a fall in blood plasma proteins from 7.53 to 4.62 or a total of 2.91 Gm. per cent within twenty-four hours following a partial bilobectomy for infection of the lung. Only 500 c.c. of blood were given at the time of operation. In view of his low protein level, 900 c.c. of additional blood were given in two transfusions which produced an elevation of his plasma protein level to 5.9 Gm. per cent by the end of six days.

In a second patient, 21 years of age, a fall from 6.94 to 5.12 or 1.82 Gm. per cent plasma proteins resulted following a lobectomy for infection of the lung. Again, only 500 c.c. of blood had been administered during the operation. After another transfusion of 500 c.c. of blood, the plasma proteins rose to 5.98 and subsequently to 6.66 Gm. per cent within the next five days.

The etiology of the marked fall in plasma proteins in these two cases were due to two factors, namely, more blood was lost during the operation than was obvious at the time, and secondly, serum loss into the wound and into the pleural space following the operation.

Only in more recent years have adequate studies been made for understanding not only the proteins of the blood, but the reserve source as well. In a review by Madden and Whipple,[3] the liver was given primary importance in the production of these substances. The authors

TABLE V

REPLACEMENT OF PLASMA PROTEIN LOSS IN PNEUMONECTOMY

DOG NO.	INITIAL WEIGHT (KG.)	INITIAL TOTAL SERUM PROTEIN	INITIAL HEMATOLOGIC FINDINGS			REPLACEMENT THERAPY (C.C.)	INFECTION PRESENT	PLEURAL FLUID REMOVED (C.C.)	LOWEST TOTAL SERUM PROTEIN (GM. %)	TIME FOLLOWING PROCEDURE (DAYS)	AMOUNT OF FALL IN P.P. (GM. %)	HEMATOLOGIC FINDINGS AT TIME OF LOWEST PLASMA PROTEIN LEVEL			DIED OR SACRIFICED	REMARKS
			HEMATOCRIT	HB.	R.B.C.M.							HEMATOCRIT	HB.	R.B.C.M.		
A. Pneumonectomy using mattress suture technique in 7 dogs																
Left pneumonectomy with plasma transfusion in 5 dogs																
366	10.9	5.64	53	16.0	8.0	200	+	440	4.90	1	0.74	53	14.5	7.6	D	Died 2 days P.O. of distemper
372	19.4	5.98	58	15.0	7.6	300	+	285	4.90	4	1.08	58	16.0	8.6	D	Died 5 days P.O. of distemper
376	10.7	7.55	59	10.5	6.0	350	+	330	6.05	2	1.50	40	10.5	5.0	D	Died 5 days P.O. of empyema
365	21.5	7.30	67	16.5	9.8	300	–	305	4.97	2	2.33	47	15.0	7.2	S	
371	18.7	6.23	56	15.0	7.5	300	+	200	5.80	2	0.43	45	13.0	6.5	D	Died 2 days P.O. with bronchial leak and empyema
Left pneumonectomy with blood transfusion in 2 dogs																
350	15.9	5.53	45	16.0	7.0	300	+	535	4.24	5	1.29	71	18.0	8.0	D	Died 6 days P.O. of empyema
349	17.2	5.60	48	14.0	5.4	200	–	240	4.76	3	0.84	60	17.5	7.0	D	Tension pneumothorax, 4 days P.O.
B. Pneumonectomy using individual ligation technique in 38 dogs																
Left pneumonectomy with serum transfusion in 6 dogs																
413	6.5	5.31	57	14.0	6.6	400	–	320	4.83	2	0.48	50	13.0	6.1	S	
432	6.4	5.14	49	13.5	5.8	400	–	205	4.94	2	0.20	46	13.0	5.6	S	
336	7.7	5.50	57	14.0	6.4	400	–	690	5.35	6	0.15	50	12.0	5.7	S	
467	15.2	5.77	61	14.5	7.2	400	–	705	5.31	6	0.46	54	13.0	6.7	S	
472	10.1	6.36	60	15.0	6.2	400	–	625	4.83	3	1.53	–	–	–	S	
Average fall in plasma proteins for 5 dogs—0.56 Gm. %																
471	15.0	6.12	44	12.0	5.8	400	+	955	5.18	5	0.77	–	–	–	D	Died 6 days P.O. of tension pneumothorax and empyema
Average fall in plasma proteins for 6 dogs—0.60 Gm. %																
Left pneumonectomy with serum intrapleurally in 24 dogs																
493	10.0	5.70	69	17.0	7.1	200	–	T*	4.41	2	1.29	64	16.0	6.6	S	
220	14.4	6.36	60	16.0	6.4	200	–	T	5.60	1	0.76	56	15.5	6.2	S	
505	6.6	6.09	58	14.0	6.2	200	–	T	4.90	4	1.19	–	–	–	S	
504B	9.3	5.88	55	14.5	5.8	200	–	T	5.64	2	0.24	–	–	–	S	
507	11.3	6.01	52	15.0	5.8	200	–	T	5.53	4	0.48	–	–	–	S	
511	12.5	5.74	65	16.0	6.8	200	–	T	5.14	8	0.60	–	–	–	S	
Average fall in plasma proteins for 6 dogs—0.76 Gm. %																

*T—for tests only.

501	15.2	5.46	69	14.5	6.5	200	+	T	4.41	4	1.05	–	–	–	D	Died of empyema 6 days P.O.
506	8.3	5.88	58	16.0	6.1	200	+	T	5.14	17	0.74	–	–	–	D	Died of empyema 18 days P.O.
510	10.3	5.53	62	14.0	5.6	200	+	T	4.31	2	1.21	–	–	–	D	Died of distemper 5 days P.O.
											Average fall in plasma proteins for 3 dogs—1.00 Gm. %					
											Average fall in plasma proteins for 9 dogs—0.84 Gm. %					
527	13.1	6.44	46	11.5	5.4	400	–	T	4.86	3	1.58	–	–	–	S	
541	13.3	5.25	56	15.0	5.9	400	–	T	5.91	2	0	–	–	–	D	Died of tension pneumothorax 7 days P.O.
									6.53	4						
546	22.2	6.05	61	16.5	6.4	400	–	T	5.70	2	0.35	–	–	–	S	
558	9.7	5.31	67	14.0	6.8	400	–	T	5.28	2	0.03	–	–	–	S	
560	20.7	6.23	71	16.0	7.4	400	–	T	5.50	4	0.73	–	–	–	D	Died of tension pneumothorax 5 days P.O.
573	9.6	5.79	61	14.0	6.4	400	–	T	5.33	1	0.46	55	13.0	5.8	S	
											Average fall in plasma proteins for 6 dogs—0.52 Gm. %					
540	9.0	4.97	68	12.5	6.1	400	+	T	5.39	2	0	–	–	–	D	Died of empyema 7 days P.O.
									6.05	4						
547	9.5	5.50	52	15.5	5.8	400	+	T	4.24	2	1.26	–	–	–	D	Died of empyema 5 days P.O.
557	15.3	5.39	51	12.0	5.6	400	+	T	4.86	1	0.53	–	–	–	D	Died of distemper 2 days P.O.
561	9.6	5.64	64	15.5	6.6	400	+	T	5.14	2	0.50	–	–	–	D	Died of distemper 3 days P.O.
331	15.1	5.74	58	15.0	6.4	400	+	T	5.31	3	0.43	–	–	–	D	Died of empyema 6 days P.O.
570	14.3	5.80	62	15.5	6.8	400	+	T	5.07	1	0.73	–	–	–	D	Died of distemper 3 days P.O.
572	8.4	5.72	56	11.0	6.2	300	–	T	4.80	12	0.92	45	12	5.8	S	
											Average fall in plasma proteins for 7 dogs—0.62 Gm. %					
											Average fall in plasma proteins for 13 dogs—0.58 Gm. %					
						Left pneumonectomy with serum intrapleurally and blood transfusion in 8 dogs										
575	10.4	5.09	60	14.0	5.9	Serum 200 c.c. I.P. Blood 100 c.c. I.V.	–	T	5.11	1	0	55	14.0	5.7	S	
									5.80	7						
581	6.7	5.98	54	15.0	5.8	"	–	T	5.25	6	0.73	58	16.0	6.4	S	
582	10.7	6.36	60	16.0	6.3	"	–	T	5.14	2	1.11	64	16.5	6.7	S	
594	10.4	5.84	59	15.5	6.1	"	–	T	5.46	6	0.38	64	16.0	6.5	S	
597	15.9	5.25	58	15.0	5.9	"	–	T	5.42	2	0	62	16.0	6.1	S	
									5.35	6						
											Average fall in plasma proteins for 5 dogs—0.44 Gm. %					
574	14.2	4.85	54	13.0	5.6	"	+	T	4.35	2	0.51	56	15.0	5.9	D	Died of empyema 4 days P.O.
576	10.2	5.65	59	15.0	6.2	"	+	T	5.11	5	0.53	63	15.5	6.4	D	Died of empyema 6 days P.O.
557	9.8	4.06	51	13.0	5.4	"	+	T	4.31	1	0	–	–	–	D	Died of distemper 2 days P.O.
											Average fall in plasma proteins for 3 dogs—0.34 Gm. %					
											Average fall in plasma proteins for 8 dogs—0.41 Gm. %					

TABLE VI

REDUCTION OF PLASMA PROTEINS BY LUNG RESECTION AND ITS REPLACEMENT IN FOUR GOATS*

GOAT NO.	INITIAL WEIGHT (KG.)	INITIAL TOTAL SERUM PROTEIN	INITIAL HEMATOLOGIC FINDINGS		REPLACEMENT THERAPY (C.C.)	INFECTION PRESENT	PLEURAL FLUID REMOVED (C.C.)	LOWEST TOTAL SERUM PROTEIN (GM. %)	TIME FOLLOWING OPERATION (DAYS)	AMOUNT OF FALL IN P.P. (GM. %)	HEMATOLOGIC FINDINGS AT TIME OF LOWEST PLASMA PROTEIN LEVEL		DIED OR SACRIFICED	REMARKS
			HEMATOCRIT	HB.							HEMATOCRIT	HB.		
1	17.5	6.01	36	14.0	0	–	75	4.48	2	1.53	30	11.0	S	Left lower lobectomy. Sacrificed 4 months P.O.
2	19.3	6.74	46	12.5	0	–	0	4.97	2	1.77	42	12.0	S	Left pneumonectomy. Sacrificed 6 months P.O.
3	33.3	6.36	46	13.0	Serum I.V. 450 c.c.	–	0	5.64	1	.72	40	12.0	S	Left pneumonectomy. Sacrificed 6 weeks P.O.
4	34.7	6.53	50	13.5	Serum I.P. 400 c.c	+	0	5.21	2	1.32	45	13.0	S	Left pneumonectomy. Small encapsulated empyema. Sacrificed 7 weeks P.O.

*Because of the scarcity of this animal as well as the increased cost as compared to dogs, only four goats were used in this problem. A definite hypoproteinemia resulted in the first two animals (control) following lung resection. The amount of plasma protein fall was materially reduced in the last two by intravenous and intrapleural administration of serum. In Goat 4 the factor of infection was present but in a very mild degree. This, added to the route as well as the amount of replacement therapy (I.P.), probably accounts for the increased fall in plasma proteins over that seen in Goat 3.

believed that there was more or less of a balance between the proteins of the blood and the body proteins—an ebb and flow or dynamic equilibrium. Madden and co-workers demonstrated in dogs that the plasma protein building material may be depleted in two to six weeks by plasmapheresis.[4] It is quite readily conceivable that a considerable depletion of reserve body proteins may take place in patients with long-standing infections, where constant suppuration is present. The rapid fall in blood plasma proteins from 7.53 to 4.62 or a total of 2.91 Gm. per cent, within twenty-four hours, in the first case cited would strongly suggest that the reserve store of proteins in this individual had been markedly lowered.

Some of the deleterious effects of hypoproteinemia have been demonstrated by animal experimentation. Thompson, Ravdin, and Frank[5] found that the normal processes of wound repair could be greatly retarded by experimentally producing hypoproteinemia in the dog. In a further investigation, Thompson, Ravdin, Rhodes, and Frank[6] used lyophil plasma in the correction of hypoproteinemia and found that the retardation in wound healing associated with lowered blood proteins in dogs could be averted by restoration of the serum protein to normal levels immediately after operation. Ravdin[7] believes that there is "no such thing as a critical level of the plasma protein at which edema becomes manifest."

The influence of hypoproteinemia on the development of postoperative infections, either in the lungs or at the site of operation must be considered. Cannon and his associates,[8] from experiments on rabbits, found a diminution of agglutins produced and increased susceptibility to infection in rabbits with hypoproteinemia as compared to their litter-mates with normal blood protein levels.

The clinical application of these data at the present time is quite obvious. In civilian practice, operations within the chest for tumor or infection should be preceded by blood or serum transfusion in the presence of a lowered blood plasma protein. Likewise, casualties on the battlefield frequently have lost considerable quantities of blood and should be carefully investigated for evidence of hypoproteinemia before making operations which in themselves would aggravate the already lowered blood proteins. Loss of additional protein during and following operation should be expected and replacement of the same planned for in advance. Not infrequently the amount of blood loss at operation is far underestimated. White and Buxton,[9] from a study of 108 operations on patients, found the average loss following various chest operations to be as follows: Thoracoplasty, 725 c.c.; lobectomy, 1607.5 c.c.; and pneumonectomy, 1458.8 c.c. The maximum loss for these operations was as high as 2895.3 c.c. These figures compare favorably with those obtained from a similar study made on patients at the University of Chicago Clinics.

In some recent publications a word of warning has appeared regarding the use of large blood transfusions. Gibbon and associates[10] demonstrated by experiments in cats that large transfusions following the resection of approximately two-thirds of the lung tissue caused death due to pulmonary edema. His experiments employed a bilateral operation and removal of a higher per cent of the total lung volume at one time than is advisable clinically. Ivy and co-workers[11] in experiments on dogs demonstrated that replacement of severe blood loss (60 per cent of blood volume) with citrated blood, if given rapidly (within five minutes), was fatal in a high percentage of animals, and cautioned against the giving of large transfusions rapidly.

These experiments, in themselves, do not preclude the use of adequate blood or plasma in replacement of blood and serum lost at operation or otherwise. However, they might tend to influence some surgeons to use smaller amounts of blood than would be advisable for the patient. In this connection it has been shown by Weech and Ling[12] that the administration of large amounts of saline solution intensified the deleterious effects normally produced at the same level of lowered plasma protein. In other words, a latent edema could be developed into severe edema by the administration of a large quantity of saline solution intravenously.

SUMMARY AND CONCLUSIONS

1. Blood plasma proteins were reduced by 33⅓ per cent to a significantly low level (approximately 4.0 to 4.25 Gm. per cent) following the resection of the lung in dogs. This reduction of the normal concentration persists for as long as ten days.

Several factors contributed to the fall in plasma proteins, namely, the removal of lung tissue, the thoracotomy wound, anesthesia, and infection.

2. Marked reduction in blood plasma proteins following resection of the lung was obviated by adequate replacement of protein loss. Replacement therapy consisted of the administration of blood or serum intravenously or serum intrapleurally. Both replacement materials and routes of administration were equally effective when the materials were given in adequate amounts.

PROTOCOL 1.

Dog 389.—Hypoproteinemia following left total pneumonectomy.

10/19/42. An 8.5 kg. mongrel had the preoperative blood studies made as seen in the accompanying summary. Under morphine 0.015 Gm. and intratracheal ether anesthesia an incision was made along the course of the left fifth interspace. After dividing the muscles along the course of their fibers, the pleural cavity was opened through the left fifth interspace. The three veins and one artery of the left lung were doubly ligated with silk and divided between ligatures. The left primary bronchus was mobilized and after closing this bronchus by three sutures (Fig. 3) the bronchus was divided. The mucosa was then cauterized with a 35 per cent solution

PROTOCOL 1

HYPOPROTEINEMIA FOLLOWING TOTAL PNEUMONECTOMY—DOG 389

Date	10/19/42	10/20/42	10/21/42	10/22/42	10/24/42	10/26/42	10/27/42	10/28/42	10/29/42	10/31/42	11/2/42	11/5/42
Weight (kg.)	8.5	—	—	8.0	—	7.7	—	—	7.2	—	—	Animal
Temperature	100.1° F.	104° F.	103.8° F.	104.2° F.	103.6° F.	104.0° F.	102.8° F.	103.2° F.	103.0° F.	102.4° F.	—	sacri-
Blood:												ficed.
Hemoglobin (Gm.)	16.5	—	—	14.0	—	—	—	14.0	—	—	—	Stump
Red cells (m.)	7.4	—	—	6.2	—	—	—	—	—	—	—	healed.
Hematocrit (%)	58	—	—	60	—	—	—	49	—	—	—	Very
Plasma protein	5.53	5.14	4.86	4.24	5.21	5.11	4.86	5.25	5.70	5.39	6.01	little
Thoracentesis:	—	Sanguin-	—	Serosan-	—	—	—	—	—	—	—	pleural
		eous		guineous								fluid
Amount (c.c.)	—	30	—	90	—	85	—	—	130	—	35	found
Hemoglobin (%)	—	25	—	0	—	30	—	—	23	—	25	
Red cells (m.)	—	2.3	—	0.3	—	1.9	—	—	2.15	—	2.0	
Hematocrit (%)	—	21	—	6	—	19	—	—	14	—	15	
Total protein (Gm. %)	—	8.36	—	4.63	—	8.12	—	—	7.58	—	—	
A. G. ratio	—	3.97	—	2.78	—	3.28	—	—	2.59	—	—	
		4.39		1.85		4.84			4.99			
Culture	—	Sterile	—	Sterile	—	Sterile	—	—	Sterile	—	Sterile	
Fluoroscopy	—	Cloudy left	—	Same	—	Fluid level (left)	—	—	Fluid		—	
Wound		No infection developed. Healed by primary intention										
General Condition		Good, continued well and took nourishment										

Left total pneumonectomy on 10/19/43. A hypoproteinemia developed and persisted for 9 days following operation.

of silver nitrate and the end closed with a running stitch of silk. The pleura of the chest wall was then irritated by massaging with a sponge (2 by 2 inches) which was left in the pleural cavity.

Closure was made using two paracostal stitches and a running stitch of silk for the intercostal structures. The muscles of the chest wall were reapproximated with continuous silk sutures, the same material being used for the subcutaneous tissues and for the skin.

With the closure of the pleural cavity, air was aspirated from the pleural space by means of a catheter brought out through the wound. When the pressures were found to be normal, the catheter was removed. The animal was returned to the kennel in good condition.

10/20/42. The animal appeared to be well, but ate little. The plasma proteins were found to have fallen from 5.53 to 5.14 Gm. per cent. The temperature was elevated.

10/21/42. Plasma proteins had fallen still further to 4.86 Gm. per cent. The general condition was good, but the dog ate little.

10/22/42. A still further reduction in plasma protein was noted to a level of 4.24 Gm. per cent and 90 c.c. of a serosanguineous fluid were removed from the pleural space. It contained a concentration of 4.63 Gm. per cent of protein.

10/26/42. Dog's plasma protein level was still below normal, but had risen to 5.11 Gm. per cent by this time. It continued below normal, as seen in the summary, for another two days and had returned to the preoperative level by the tenth day following operation.

Fluoroscopic and x-ray examinations made on Oct. 20, 22, 25, and 29 revealed fluid in the left pleural space. Aspiration of this fluid on Oct. 20, 22, 26, and 29 revealed no evidence of infection on culture. The wound healed by primary intention.

The animal was sacrificed on 11/5/42, seventeen days following operation. The bronchial stump was solidly healed and little pleural fluid remained. As shown by the summary for this animal, a hypoproteinemia of a considerable degree developed. It was seen to begin by the first day following operation and persisted for ten days, being most marked on the third day.

PROTOCOL 2.

DOG 575.—Replacement of blood protein loss following left total pneumonectomy.

5/3/43. A 10.4 kg. mongrel had preoperative blood studies made as shown in the accompanying summary. Under ether anesthesia, preceded by morphine, an incision was made over the course of the left fifth interspace. The muscles were divided in the direction of their fibers and the pleural cavity entered through the left fifth interspace. The three veins and one artery of the left lung were mobilized, doubly ligated with silk, and divided between ligatures. The left primary bronchus was mobilized and three silk sutures applied as illustrated in Fig. 3. The bronchus was then divided and the mucosa cauterized with a 35 per cent solution of silver nitrate. The end of the bronchus was then closed with a running stitch of silk. The pleura of the chest wall was then irritated by massaging with a gauze sponge (2 by 2 inches) which was then left in the pleural cavity. The chest wound was closed using two paracostal sutures of silk and a running stitch of silk for the intercostal structures. The muscles of the chest wall were then approximated with silk sutures and the same material was used for the subcutaneous tissues and skin. As the wound was being closed, air was removed from the pleural space and replaced with 180 c.c. of serum, the intrapleural pressure being normal when the catheter used for this was removed. Following this, a transfusion of 100 c.c. of whole blood was given. The animal was returned to the kennel in good condition.

PROTOCOL 2

REPLACEMENT OF BLOOD PROTEIN LOSS FOLLOWING TOTAL PNEUMONECTOMY—DOG 575

Date	5/3/43	5/4/43	5/5/43	5/6/43	5/7/43	5/8/43	5/11/43	5/18/43
Weight	10.4	—	—	10.8	—	—	—	10.3
Temperature	100.4° F.	103.2° F.	102.8° F.	103.2° F.	103.8° F.	103.6° F.	103.8° F.	—
Blood								
Hemoglobin (Gm.)	14	—	—	14	—	—	14	—
Red cells (m.)	5.9	—	—	5.7	—	—	5.7	—
Hematocrit (%)	58	—	—	55	—	—	55	—
Plasma protein	5.09	5.30	—	5.46	5.29	5.74	5.80	—
Thoracentesis	—	—	for culture	—	for culture	20 c.c. removed	—	—
Culture	—	—	sterile	—	sterile	sterile	—	—
Fluoroscopy	—	Fluid on left side only	—	—	Some shift of the heart to the right	Same	No shift of mediastinum	—
Wound	Healing by primary intention							
General condition	Remained good							

Left total pneumonectomy on 5/3/43. Dog received 180 c.c. of serum intrapleurally and 100 c.c. of blood intravenously following operation. When sacrificed two weeks later, 100 c.c. of serum were found in the left pleural space.

5/4/43. The animal was in good condition, but ate little. The plasma proteins had risen slightly from the preoperative level.

5/8/43. Plasma proteins continued to rise and the fluid removed from the pleural space remained sterile on culture. The wound healed by primary intention and the dog's general condition remained good. The preoperative level of plasma proteins was a low normal and by eight days following operation had risen 0.75 Gm. per cent.

5/18/43. The animal was sacrificed. The bronchial stump was found to be solidly healed. Approximately 100 c.c. of serum were found in the left pleural space.

We wish to thank Mr. Magnus Harding and Miss Nancy Corbin for technical assistance and protein determinations made in this study.

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ON THE MANAGEMENT OF BURNS

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BURNS are the trade-marks of war. Flaming arrows, the boiling pitch and tar pots, and even the modern flame throwers are practical products of war. But direct attempts to produce thermal injury are secondary in importance to the burns that result from bombings, and the accidents, including civilian disasters, that are coincidental with war.

Recently released statistics reveal that 60 per cent of the casualties at Pearl Harbor were the result of burns. Slightly lower percentages are reflected in the statistics available following the bombings of Britain. And yet, in spite of a clearer understanding of the pathologic effect of burns, the percentage of deaths from burns has not been appreciably reduced in the past years. Those patients suffering from burns fall into two broad classes, the quick, and the dead. The quick survive primarily because of the presence of enough intact skin compatible with life. From a practical viewpoint, there are but two types of burns, the third degree burns and the remainder. The only factors that enter into any burn are the extent and the degree. The extent of "the remainder" may cause them to fall into the "third degree type" from a standpoint of therapeusis, for in truth, the only type of lesion which presents a real test is the third degree burn. Because of this fact, the adequate management of burns revolves about two main trends of thought—the general treatment of the patient and the local treatment of the burned area. The primary object of this discussion is to emphasize further the primal importance of the first of these tenets—the general treatment of the patient—and to sublimate the local treatment of the burned area, even to ignore it to such an extent that one can say that almost any type of local treatment which will do no further harm will suffice in first degree and second degree burns, if they are not too extensive, and ultimate recovery will result not because of the local treatment, but in spite of it.

It is an accepted fact that from 60 to 75 per cent of all burn patients that die, do so because of peripheral vascular failure. In treating patients with burns it is not necessary to differentiate between primary and secondary shock since the only clinical difference between burn shock and that due to other causes is in the degree of immediate hemoconcentration. Although the classical appearance of the patient in

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"shock" in the terminal phases of many diseases is accepted as a normal occurrence as the result of a sequence of pathologic events, the use of this term shock, actually indefinable in itself, has served to picture a clinical entity rather than indicate its mechanism, particularly following injury. It has been apparent since the days of Harvey that an adequate circulation of blood is dependent upon a proper output from the heart. This cardiac output obviously may be limited by an increase in the vascular bed or by a diminution in the volume of the blood. Adequate experimental evidence is at hand to show conclusively the sequence of events in the production of shock following burns. A burn produces local capillary injury with an immediate loss of some of the components of the blood, chiefly plasmalike substances in the tissues at the site of the burn. There is, in addition, loss of plasma from the injured skin surfaces. These two factors alone account for a definite loss in total blood volume. When this loss becomes sufficient—and this loss is immediate and quantitatively rather great—to be incompatible with the maintenance of an adequate circulation, the resultant anoxemia of the tissues throughout the body per se produces an increase in the permeability of the capillary walls. A general loss of electrolytes, water, and protein from the blood is inevitable and completes the vicious cycle. The signs of peripheral vascular failure, the vasoconstriction as evidenced by the ashen or slightly cyanotic skin, the profuse sweating, and the hyperpnea are merely evidences of the sole compensatory mechanism the body possesses to offset this lack of adequate total blood volume. The emergency nature of this impending condition must be emphasized. The need for prompt and adequate treatment to offset this condition before irreparable anoxemic tissue damage occurs and pathologic changes in the viscera are established, that are irreversible once established, is essential, imperative, and requires true clinical "teamwork."

Therapeusis is directed primarily toward the two outstanding pathologic conditions that present themselves in every severe burn—the lack of plasma in the circulation reflected in inadequate blood volume and the generalized anoxemia of the tissues.

Certain elementary general therapeutic measures are not utilized to their fullest extent in many instances. The immediate use of morphine in all conscious patients is clearly indicated and should be given in large enough amounts to produce its physiologic effect. The correct answer to "how much?" is "enough" and the two "yardsticks" are the freedom from pain and the respiratory rate. In the presence of pain, morphine should be given in ¼ gr. doses, until the respiratory rate drops to 12 per minute. The adequate administration of morphine is the first step toward the production of rest, an all-important phase in offsetting impending "shock." This should be followed immediately by physical immobilization of the burned parts. That the primary function of all dressings regardless of the type of injury is that of

providing physiologic rest is an accepted fact; attention is directed to the rest that results from immobilization of a traumatized area, be it thermal in origin or not. Too much stress cannot be placed upon the immediate local treatment of burns as an integral part of the general treatment. This immobilization to prevent further shock can only be brought about through the use of adequate dressings using cotton, mechanics waste, folded newspapers, and even splints to provide the necessary bulk. While "rest" is thus being brought about, adequate provision for the control of the loss of body heat must be considered as the second feature in the general treatment of burns. Warmth, but not heat, is essential. The skin is a most important organ and its chief function is the major role it plays in the control of the loss of body heat. Clinical evidence of this often overlooked factor is the persistent subnormal temperatures of patients upon admission that have suffered major losses of skin surface. It is imperative that normal body heat be retained and maintained at all times to avoid impending shock. Climatic conditions obviously offset the control of this phase of treatment; hot climates obviously differ from the cold of the polar regions. The presence of bulky heat-retaining dressings in the tropics must be occasionally counteracted by transporting the patients to a cold room to prevent hyperthermia.

The most outstanding clinical feature of shock is circulatory collapse. Since the transport of oxygen is the prime function of the circulation, failure of this mechanism increases the tissue anoxemia and a vicious cycle is established. Until the cause of the shock can be removed and the blood volume restored, oxygen is the best, the only way, of combating anoxemia. To withhold this specific treatment until respiratory and tissue decompensation are apparent is a most common therapeutic error. As a specific measure, the use of oxygen to prevent tissue anoxemia and the resultant spread of the peripheral vascular failure cannot be minimized. For example, all doctors appreciate the fatal effect of even temporary cerebral anoxemia when respirations fail regardless of cause and, in this instance, artificial respiration is invariably started at once. An exact parallelism exists in burn shock. Impending tissue anoxemia is always present, which if allowed to persist will ultimately result in an increase in the capillary permeability and deepen the degree of the shock. The normal percentage of alveolar oxygen can be doubled by the administration of oxygen through a nasal catheter at the rate of 1½ L. per minute, tripled and even quadrupled by placing the patient in an oxygen tent, and increased some five times by the administration of oxygen by means of a B-L-B mask. Obviously, the more extensive the burn, the higher the percentage of alveolar oxygen required to prevent tissue anoxemia. It must be stated, however, that although the best and most economical method of administering oxygen is by means of a B-L-B mask, this method of choice is not practical unless the patient is able to understand and cooperate,

or is wholly unconscious. Experience has shown that the struggles of the irrational, uncooperative patient while using a B-L-B mask may do more ultimate harm than the good that comes from the administration of oxygen.

The most important single step in the treatment of burns is the intravenous administration of plasma to restore the normal total blood volume. The use of plasma in each instance must be quantitative and individualized for each burned patient. To calculate the dosage of plasma, one of several simple formulas may be used. The clinical method provides for the administration of plasma in sufficient quantities to maintain adequate peripheral circulation, as shown by testing the bleeding time from a needle prick of the ear. This method is *always* available. The first-aid formula of Berkow advocates the use of 50 c.c. of plasma for each per cent of the body surface involved in a deep burn. When adequate laboratory facilities are available, the hematocrit method as suggested by Harkins has proved most valuable and in addition gives one a yardstick as to the progress of the individual case. This method calls for the administration of 100 c.c. of plasma for every point exceeding 45, the normal hematocrit for adults, except when the total proteins fall below 600 mg., when an additional 25 per cent of plasma must be given to cover this deficit.

It is not unusual for many of the major peripheral veins to be thrombosed as the result of burns. Thermal injury to the median antecubital fossae, the ankles, and the hands is most distressing and common, but the jugular and scalp veins are frequently not involved. The sternum is always available. The intravenous administration of plasma is extremely trying on the veins themselves and thrombosis eventually results. The rate of flow of plasma should be relatively slow but continuous. The use of a large bore needle and a three-way by-pass stopcock to allow continuous intravenous administration of plasma in the severe cases is ideal.

Two crystalloids, the chlorides and the carbohydrates, must be given to all patients suffering from burns. It has been shown that 10 Gm. of chloride per twenty-four hours will keep the average adult in chloride balance. Enough carbohydrate in the form of sugar must be given to protect the liver against undue depletion of the liver glycogen. It is absurd to use the few remaining superficial veins that are so essential for the administration of plasma for the parenteral administration of either chlorides or carbohydrate. The installation of a nasal catheter for feeding and for the administration of both chlorides and carbohydrates is essential. A No. 16 soft rubber urinary catheter has the physical advantages of hanging free in the esophagus; the presence of its normal funnel-shaped tip easily admits the entrance of a cork to block the regurgitation and acts as a buttress for the adhesive to hold it in place. In this fashion a calculated high protein diet containing

vitamins, electrolytes, etc., is assured at all times as well as an adequate caloric and fluid intake without resorting to parenteral therapy.

Diet should be high protein in character from the onset as there is an abundance of experimental and clinical evidence that focal necrosis of the liver will result from a protein-free diet just as easily as from continuous lowered serum protein levels of the blood.

Actually the administration of these crystalloids by the intravenous route (the "popular" route can produce irreparable damage) merely "washes out" the plasma from the total blood volume at an alarming rate and should not be employed.

The local treatment is directed at the immobilization of the burned parts to prevent further shock, the prevention of sepsis, the prevention of contractures, and the assistance in epithelization.

Most of the patients with burns who die after a ten-day survival, die from sepsis. The use of 2 Gm. of sulfadiazine as soon as practical and repeated in eight hours followed by 1 Gm. every six hours has its proponents who insist that the blood level of 7.5 mg. per cent should be maintained for the first two weeks. Contractures are prevented only by early skin grafting. Splinting will not prevent contractures nor will the application of plaster casts alter this pathologic result of burns. Such procedures only mask the true situation and all treatment should be directed toward the preparation of the area for grafting at the earliest possible moment to prevent this unfortunate complication. Application of pressure dressings remains the best known procedure irrespective of the application of many so-called epithelial stimulants such as scarlet red to encourage epithelial spread and regeneration. All burn cases are candidates for tetanus and must be treated accordingly. The local use of tannic acid sprays, the various aniline dyes, the sulfonamides, the tub baths, and the Bunyan envelopes, although staunchly defended by their respective proponents, minor factors and may be dismissed without further comment. The use of fine mesh xeroform gauze held in place with firm and bulky dressings is my method of choice in handling these local trivia.

The most serious complication is the presence of fractures and in a severely burned individual this complex taxes one's ingenuity and must be dealt with accordingly in an individualized fashion.

Tracheotomies in those patients who have inhaled flames are essential and lifesaving. If there is any question as to the need for this operation, it should be carried out without delay. The time to perform a tracheotomy is before it becomes urgent from the clinical signs.

An ophthalmologist must check the eyes of all patients burned about the face as soon as practical following admission, confirm his findings by a second examination within twelve hours, and include them in the record.

Special nurses who not only assist in the administration of plasma and the local care of the wounds but who are trained to pay particular

attention to the intact skin are invaluable. Similarily, the roles of the neuropsychiatrist and the Red Cross worker cannot be minimized. Inasmuch as burns rank close to the common cold and fractures from a standpoint of hospital days, these seemingly endless days must be planned in a thoughtful fashion for the patient convalescing from burns.

Recent experimental evidence indicates that barbiturates act as a specific in the prevention of shock in burn cases, but there are no clinical data at hand to substantiate this fact at this writing. In a similar vein, the use of adrenal cortex has its advocates. Rhoades, Wolff, and Lee have experimental evidence to show its efficacy. At present this is a nonstandard drug and is not available.

In conclusion, let it be stated that the principles of the treatment must receive primary consideration in the management of severe burns and one's attention must not be distracted by the employment of local "specifics" classed by some as "trivia."

This apologia represents the reflections obtained from a year's experience with burns in the Middle East without any recourse to the current literature which must abound with notes on this all-important subject.

SECONDARY CARCINOMA OF THE MANDIBLE

An Analysis of Seventy-one Cases*

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THE likelihood of success in treating secondary carcinoma of the mandible is not as infrequent as one might think from isolated experiences, or from the literature.[1] Secondary neoplasms of bone are comparatively frequent,[2, 3, 4] as many primary tumors are responsible for bone involvement; the incidence of metastasis to the mandible appears to be rare, as few references[1, 5-9] to metastatic carcinoma of the mandible are noted in the literature. Stern and Shephard[10] summarized from the literature fifty-five cases of metastatic involvement of the jaw, and reported one case of metastasis to the mandible from a bronchiogenic carcinoma.

The following analysis of seventy-one case records of secondary invasive carcinoma of the mandible treated at the University of Minnesota Hospitals and the Tumor Clinic,[11] from 1924 to 1943, include malignancies secondary to primary carcinoma of the lip, buccal mucosa, lower alveolar process (gingiva), and the floor of the mouth. This study was not undertaken as a basis for any broad conclusions, but rather to ascertain the results of methods employed by surgeons at the University of Minnesota Hospitals.†

GENERAL CONSIDERATIONS

Age.—The youngest patient in this group was 48 years of age and the oldest 84. Twenty-nine per cent were in the sixth decade and 30 per cent in the seventh.

Sex.—There were eight women and sixty-three men. The average age of the men was 65 years and of the women 63.

Stage of Disease.—The cases were studied as a group of seventy-one patients and divided as to the topographical location of the initial lesion and classified according to the extent or stage of the disease as suggested by Howes and Bernstein.[12] Stage I: A lesion 1.5 cm. or less in diameter and confined to its local anatomic structure as far as can be determined by palpation and microscopic examination at the time of treatment. Stage II: A lesion over 1.5 cm. in diameter, still limited to the local

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*Data were compiled by the author prior to active military service.

†The operations reported herein were performed by numerous surgeons; the chief responsibility for the care of these cases devolved successively upon Doctors W. T. Peyton, J. R. Paine, and latterly myself. Currently, Dr. Clarence Dennis is supervising the care of these cases in the surgical clinic.

area. Stage III: A lesion with local extension but no evidence of regional metastases, that is from lip to gingiva and mandible, from buccal mucosa to gingiva and mandible. Stage IV: A lesion with local extension and regional lymph node metastases confirmed by histologic examination.

TABLE I

CLASSIFICATION OF THE PRIMARY LESIONS ACCORDING TO LOCATION AND STAGE OF INVOLVEMENT

STAGE	LIP	BUCCAL MUCOSA	GINGIVA	FLOOR OF THE MOUTH
I. Localized lesion up to 1.5 cm. in diameter	8	1	4	2
II. Larger than 1.5 cm. without local extension	9	9	10	1
III. Localized extension with bone invasion	0	2	11	0
IV. Local extension and lymph node metastases	0	1	11	2

Symptoms.—More than half of these people were unaware of an oral lesion of a serious nature until pain, ulceration, hemorrhage, or metastases as a submandibular swelling was experienced. In the clinical Stage III cases, mandibular bone pain, or toothache, was the principal complaint which obliged the patient to seek medical or dental attention.

Etiologic Factors.—Chronic intraoral irritation in some form was found in all of the group. Oral sepsis, mild or severe, and dental caries were present to a degree in most of the patients. Beiswanger[13] reported 95 per cent of 160 cases of oral carcinoma associated with dental sepsis. Ill-fitting dentures of five patients were thought to be directly responsible for their gingival carcinoma. Two men were known to have carried tobacco quid for years at the site of a buccal lesion. Intraoral leucoplakia was recorded as a precancerous lesion in eight (11 per cent) of the patients. Complete data were lacking in many of the charts, and it is possible that the occurrence of all etiologic factors was considerably larger.

Approximately 40 per cent of the patients in this group had been edentulous for years, and in many instances attributed their disaster to earlier dental extractions. Malassez[14] pointed out that epithelial cell rests of the periodontal membrane, when stimulated by inflammation, infection, or trauma, were responsible for the development of radicular cysts in the edentate as well as the dentate jaw. Thoma[15] reported a carcinoma of the mandible, grade II, arising from the peridental membrane at the apex of a third molar and editorially states, "A small amount of membrane attached to an extracted tooth may be just a remnant of the dental follicle, but it may also be a new growth; adamentinoma may form in the young, or carcinoma may form after middle age from remnants of tooth germs."

Delay in Diagnosis and Treatment.—Delays were observed with consistent frequency as in other malignancies.[16] Smith[17] determined an average delay of eight months for oral carcinoma, and Martin and Sugarbaker[18] determined a delay of five months in patients with carcinoma of the floor of the mouth.

In this series the patient endured a delay of several months prior to diagnosis or appropriate treatment. There were seventeen patients with lip cancer, which averaged a delay of 12.4 months before treatment; eleven patients with carcinoma of the buccal mucosa with a delay of 13.5 months; twenty-one patients with gingival lesions averaged a delay of 9.5 months, and two patients with cancer of the floor of the mouth suffered a delay of 6.5 months. The average delay for fifty-one cases was 8.4 months.

Responsibility for Delay.—The fault lies jointly with the physician and the patient. The common mistake of the patient was his unexplained procrastination. He may have suspected a serious condition, but delayed investigation until pain or disability forced him to seek medical care. A further serious error was a 10 per cent incidence of patients accepting cancer therapy from unapproved sources, and unfortunately an equal number of cases received unacceptable medical treatment.

The physician's responsibility for delay in this group of patients with oral carcinoma was often shared by the dental surgeon. The most common cause for delay in instituting proper treatment was the failure of both the dentist and the physician to suspect a malignant lesion. Only 10 per cent of the patients were promptly referred by the dentist or physician to an appropriate source of therapy.

Differential Diagnosis.—In the present study there was no real problem of differential diagnosis because all the lesions were biopsied and examination of the tissues revealed carcinoma of squamous cell origin. However, in fifteen instances an opportunity to establish an early diagnosis was overlooked as eleven patients had had extractions of teeth in the presence of intraoral carcinoma; the dentures of two patients had been revised to accommodate the growth of a tumor, and one patient was given prolonged treatment for a Vincent's infection.

Gettinger's[19] illustrative account stressed the importance of cooperation between individuals responsible for the diagnosis of intraoral lesions and the pathologist. Darlington,[20] in an analysis of 1,150 specimens of tissue removed for histologic examination, found 474 tumors; 227 epuli and polyps, 158 radicular cysts and granulomas, 24 dentigerous cysts, 12 ameloblastomas, and 53 carcinomas.

CARCINOMA OF THE LIP

There were seventeen patients in this group and all had their primary growth on the lower lip. At the time of the initial treatment which

consisted of surgical excision or radiation, or both, eight presented Stage I lesions, and nine had Stage II growths. Seven Stage I lesions were controlled for an average of 4.3 years, and four of these for the duration of the disease. Four men with primary Stage II disease, uncontrolled by treatment, returned with Stage IV involvement; one, after a duration of four years, presented an inoperable growth, and three, after two, seven, and nine years of temporary control, refused surgical treatment. Six Stage II growths were controlled for an average of 4.6 years, and two were controlled permanently.

TABLE II

CLINICAL SUMMARY OF THIRTEEN PATIENTS SUBJECTED TO PARTIAL MANDIBULAR RESECTION

(Primary Carcinoma of the Lip)

Case number	1	2	3	4	5	6	7	8	9*	10	11	12	13
Age	84	80	76	64	59	58	54	79	60	66	52	51	50
Stage of initial lesion	I	I	I	I	I	I	I	II	II	II	II	II	II
Treatment of primary	S-R	R	R	R	S-R	R	S	R-S	S	S	S	R	S-R
Years primary controlled	5	3	2	6	4	5	5	1	3	13	2	5	3
Stage at time of resection	III	III	IV	IV	IV	IV	IV	IV	IV	III	IV	IV	IV
Resection years after onset	2	3	2.5	5.5	4	5	5	1	3	5	2	3	3
Tracheotomy	+	–	–	–	+	+	+	–	–	–	–	–	–
Mandibular resection	+	–	–	–	–	–	+	+	–	+	–	+	+
Hemisection and disarticulation	–	+	+	+	+	+	–	–	+	–	+	–	–
Suprahyoid dissection	+	–	+	+	+	+	–	–	–	–	–	+	–
Supra-omohyoid dissection	–	+	–	–	–	–	–	–	+	–	+	+	–
Oral cutaneous fistula	+	+	+	+	+	–	+	–	+	+	+	–	–
Postoperative radiation	–	–	+	+	+	+	+	–	–	–	–	+	–
Postoperative death	–	+	–	–	–	–	–	–	–	–	–	–	†
Alive free of disease (yr.)	3†	.	.	.	.	.	.	.	2	.	.	.	.
Dead free of disease (yr.)	.	.	.7	.	.	.	.	.	T	8	.	.	.
Alive with disease (yr.)	.	.	.	.	.	.	.	.	T	.	.	.	.
Dead with disease (yr.)	.	.	.	.5	.2	1.5	1	1	T	.	.	2	.

*Female.

†Last follow-up record of patients completed February, 1943.

T, Patient untraced.

Thirteen patients (Table II) accepted a one-stage partial mandibulectomy which varied in extent from segmental mandibular resection with a suprahyoid neck dissection to a hemisection and disarticulation of the mandible with a supra-omohyoid dissection and sacrifice of large portions of the cheek and lips. Three patients returned two, three, and five years after initial treatment, with Stage III carcinoma, and ten returned one to five years after initial therapy, with Stage IV disease.

CARCINOMA OF THE BUCCAL MUCOSA

There were thirteen patients in this group. One Stage I lesion was controlled for one year, while nine Stage II lesions were arrested for

an average of 3.9 years; two were permanently controlled. All of these patients were operated upon for secondary carcinoma of the mandible, except a 62-year-old woman who presented an inoperable Stage IV growth with extensive metastases which was first noticed nine months previously as a "canker sore." Palliative radiation was given, with death occurring two years after the onset of the disease.

One patient, aged 57 years (Table III) presented a Stage II carcinoma treated by radiation and surgery, was well for sixteen years, then suffered a recurrence which was treated by a hemisection of the mandible and disarticulation with a supra-omohyoid cervical lymph gland dissection and postoperative radiation therapy. At the time of the last follow-up (February, 1943) he was alive, with a recurrence.

TABLE III

CLINICAL SUMMARY OF TWELVE PATIENTS SUBJECTED TO PARTIAL MANDIBULAR RESECTION

(Primary Carcinoma of the Buccal Mucosa)

Case number	1	2	3	4	5	6	7	8	9	10	11	12
Age	53	75	74	71	70	65	64	57	50	48	54	70
Stage of initial lesion	I	II	II	II	II	II	II	II	II	II	III	III
Treatment of primary	R	S-R	S-R	S	S-R	S-R	S-R	R-S	R	R	S	S
Years primary controlled	1	3	1	2	1	.5	2	18	2	1	15	.5
Stage at time of resection	III	IV	IV	IV	III	III	III	IV	III	III	III	III
Resection years after onset	1	5	7.5	5	1	1	5	16	1	1	.5	1
Tracheotomy	+	+	+	+	−	−	−	+	−	−	−	−
Segmental resection of mandible	−	−	+	−	+	−	+	−	−	+	−	+
Hemisection and disarticulation	+	+	−	+	−	+	−	+	+	−	+	−
Suprahyoid dissection	+	+	+	+	+	−	−	+	+	+	−	−
Supra-omohyoid dissection	−	−	−	−	−	+	−	−	−	−	+	−
Oral cutaneous fistula	+	+	−	−	+	+	+	+	+	−	+	+
Postoperative radiation	+	+	+	+	−	+	−	+	+	−	−	−
Postoperative death	−	−	−	−	−	−	−	−	−	+	−	−
Alive free of disease (yr.)	4	.7	.	.5	9	.	4	.	3	.	.	.
Dead free of disease (yr.)	T	T	.	.	.	14	T	.	.	.	.	.
Alive with disease (yr.)	T	T	.	.	.	.	T	2	.	.	17	.
Dead with disease (yr.)	T	T	.3	.	.	.	T	.	.	.	.	1

T, Patient untraced.

Two patients presenting primary buccal lesions with bone invasion (Stage III) were treated by surgery; one with cautery excision and segmental resection of the mandible succumbed to the disease after a year; the other patient was submitted to a radical extirpation of the growth by means of hemisection and disarticulation of the mandible with a simultaneous unilateral supra-omohyoid dissection. He remained free of disease for fifteen years, then returned with a recurrence in the cervical skin flap employed to close the defect in the full thickness of the cheek incurred at the time of the original operation.

CARCINOMA OF THE GINGIVA AND ALVEOLAR PROCESS

In this group there were thirty-six cases, five of them were found to be inoperable and three patients refused surgical intervention. There

were three Stage I lesions controlled for two to four years, and eight Stage II cancers controlled for an average of 15.5 months. The trend of treatment in this small group indicates that the Stage II lesions were inadequately treated, either by surgery, radiation, or both. More extensive surgery, as shown by results of the treatment of the secondary group (Table IV), probably would have initially controlled a higher percentage of the entire group of Stage II cancers.

TABLE IV

CLINICAL SUMMARY OF ELEVEN PATIENTS SUBJECTED TO PARTIAL MANDIBULAR RESECTION

(Primary Carcinoma of the Lower Gingiva, Stages I and II)

Case number	1	2	3	4	5	6	7	8	9	10	11
Age	63	56	50	72	72	66	65	64	64	64	31
Stage of initial lesion	I	I	1	II	II	II	II	II	II	II	II
Treatment of primary	R	R	S	R	S-R	S	S-R	R	S-R	R	R
Years primary controlled	2	4	2	2.5	1	2	.3	1.5	1	1	1
Stage at time of resection	IV	IV	IV	IV	IV	III	IV	IV	III	III	III
Resection years after onset	2	4	3	2.5	1	2	1.5	1.5	1	1	1
Tracheotomy	–	–	+	+	–	–	+	–	–	–	–
Segmental resection of mandible	+	+	+	+	–	+	+	–	–	+	+
Hemisection and disarticulation	–	–	–	–	+	–	–	+	+	–	–
Suprahyoid dissection	+	+	+	–	–	+	+	–	–	+	+
Supra-omohyoid dissection	–	–	–	+	–	–	–	+	+	–	–
Oral cutaneous fistula	–	–	+	+	–	–	+	+	–	–	–
Postoperative radiation	–	–	–	–	–	–	–	–	+	+	–
Postoperative death	–	–	–	–	–	–	–	+	–	–	–
Alive free of disease (yr.)	.	6	I	.	1	2	.5	.	16	6	4
Dead free of disease (yr.)	4	T	T	.7	T	T	.	.	.	.	T
Alive with disease (yr.)	.	T	T	.	T	T	.	.	.	.	T
Dead with disease (yr.)	.	T	T	.	T	T	.	.	.	.	T

T, Patient untraced.

There were seventeen advanced carcinomas of the lower gingiva, eleven Stage III, and six Stage IV. There was no correlation of size with duration or with extension to adjacent structures. Five of these lesions (Table V) were treated by intensive radiation prior to jaw resection.

CARCINOMA OF THE FLOOR OF THE MOUTH

There were five cases with primary lesions in the floor of the mouth. Three lesions, two Stage I and one Stage II, were controlled for six months, and one Stage IV lesion was initially treated two years after onset by radiation and surgery (Table VI).

One patient, admitted with a Stage IV involvement, was a model of unacceptable therapy. He complained of a "canker sore" in the right anterior third of the floor of the mouth which had been present for a year. Initial treatment consisted of inadequate surgical diathermy excision. (In this connection, the accounts of Eggers[21] and Ochsner[22] may profitably be reviewed by those who occasionally must perform radical operations for cancer.) There was a prompt recurrence, treated with deep x-ray therapy, which could not be completed because of pain. Narcotic addiction quickly developed. In a few months a unilateral supra-

TABLE V

CLINICAL SUMMARY OF SEVENTEEN PATIENTS SUBJECTED TO PARTIAL MANDIBULAR RESECTION

(Primary Carcinoma of the Lower Gingiva, Stages III and IV)

Case number	1	2	3	4	5	6	7	8	9	10	11*	12	13	14	15	16*	17*
Age	75	74	71	70	69	68	61	59	56	54	50	76	70	67	67	64	61
Stage of initial lesion	III	III	III	III	III	III	III	III	III	III	III	IV	IV	IV	IV	IV	IV
Treatment of primary	R-S	S-R	R-S	S	S	S	S	S	S	R-S	R-S	S	R-S	S	S	S	S
Stage at time of resection	III	IV	III	III	III	III	III	III	III	III	IV	IV	IV	IV	IV	IV	IV
Tracheotomy	–	–	–	–	+	–	–	–	–	–	+	+	–	–	–	–	–
Segmental resection mandible	+	–	–	–	–	–	–	+	+	+	+	–	+	–	–	–	–
Hemisection and disarticulation	–	+	+	+	+	+	+	–	–	–	–	+	–	+	+	+	+
Suprahyoid dissection	–	+	+	+	+	–	–	+	–	–	–	+	+	+	–	–	–
Supra-omohyoid dissection	–	–	–	–	+	+	–	–	+	+	–	–	–	–	–	+	–
Oral cutaneous fistula	–	+	–	–	+	+	–	–	–	–	+	+	–	–	–	–	–
Postoperative radiation	–	–	–	–	+	–	–	–	–	–	–	–	–	–	–	+	–
Postoperative death	–	–	+	–	–	–	+	+	–	–	–	–	–	–	+	–	+
Alive free of disease (yr.)	.	.2	.	7	.	.8	.	.	.	3	.	.	.2	.2	.	.1	.
Dead free of disease (yr.)	8		.	.	.	.	.	.	.	.	.	.2	.	.	.	.	.
Alive with disease (yr.)	.		.	.	.6	.	.	.	.	.	.	.	.	.	.	.	.
Dead with disease (yr.)	.	.	.	.	.	.	.	.	1.5	.	.8	.	.	.	.	.	.

*Female.

TABLE VI

CLINICAL SUMMARY OF FOUR PATIENTS SUBJECTED TO PARTIAL MANDIBULAR RESECTION

(Carcinoma of the Floor of Mouth)

Case number	1	2	3	4
Age	76	54	65	82
Stage of initial lesion	I	I	II	IV
Treatment of primary	R-S	S-R	R-S	R-S
Years primary controlled	1	.5	.5	
Stage at time of resection	IV	IV	III	IV
Resection years after onset	2	2	1	2
Tracheotomy	–	–	–	+
Segmental resection of mandible	+	+	+	+
Hemisection and disarticulation	–	–	–	–
Suprahyoid dissection	+	+	+	–
Supra-omohyoid dissection	–	–	–	–
Oral cutaneous fistula	+	+	–	–
Postoperative radiation	+	+	–	–
Postoperative death	–	–	–	+
Alive free of disease—(yr.)	.	2	.	.
Dead free of disease (yr.)	.	.	.	.
Alive with disease (yr.)	.	.	.7	.
Dead with disease (yr.)	1	.	.	.

hyoid neck dissection was performed for metastatic cervical carcinoma, without the control of the primary growth, which fails to meet one of the fundamental criteria of operability recommended by Duffy.[23] A number of teeth were extracted for pain in the mandible some time after the radiation therapy.[24] The patient refused surgical treatment for a Stage III lesion, and some months later accepted a tractotomy which provided relief from pain up to the time of his death approximately twenty-six months after the onset of the disease. Owing to the malignant character of carcinoma of the floor of the mouth, prompt and effective initial therapy is a prime requisite, as any delay or inadequate therapy carries a grave responsibility.

MANDIBULAR RESECTION

The essential surgical considerations of partial mandibular resection, combined with dissection of the submandibular triangles, were fully described in the papers of Bartlett and Callander,[25] Blair and Brown,[26] Crile,[27] Hotchkiss,[28] Kennedy,[29] Padgett,[30] Polya,[31] Simmons,[32] Taylor and Nathanson,[33] Warren,[34] and others.

Fifty-seven patients with secondary carcinoma of the mandible were subjected to mandibular resection, with ten operative deaths (17 per cent). There were thirteen jaw resections for secondary carcinoma due to primary carcinoma of the lip, with three deaths; twelve operations for secondary carcinoma due to primary carcinoma of the buccal mucosa, with one death; twenty-eight operations for secondary carcinoma due to primary carcinoma of the lower gingiva, with five deaths; and four resections for secondary carcinoma due to carcinoma of the floor of the

mouth, with one death. The deaths were due to sepsis, pneumonia, and respiratory obstruction. The cause of one death was not clear from the record.

Seven cases presented an associated radionecrosis; and although presence of cancer was not substantiated by the histopathologic examination of four of them, it was thought that as a result of the usual practice of sectioning only a reasonable portion of the operative specimen of the mandible and contiguous structures the microscopic presence of cancer in the bone could easily have escaped detection, or could have been destroyed by the irradiation reaction.

Wangensteen has advocated in this clinic the use of tracheotomy in extensive resections of the mandible, as a means of controlling immediate postoperative mortality. In the last fifteen resections done by me, routine preliminary tracheotomy was performed, subsequently free of all complications and well tolerated by the patients. This procedure delayed the principal operation about five days, but it was found on several occasions to be a valuable part of the planned procedure, especially in those cases in which supporting structures of the base of the tongue had to be removed. In addition to providing a constant airway, it provides an opportunity for the anesthetist to use his equipment removed from the field of operation. General anesthesia was employed in these operations, and accomplished with intratracheal administration of oxygen and ether, except recently when pentothal sodium was used intravenously and the airway maintained through the tracheotomy tube.

The treatment of extensive carcinoma involving the mandible presents an opportunity to direct surgical procedures toward the treatment of the primary lesion and the lymph nodes metastases as a single procedure. The extent of the resection and of the dissection was generally planned to include one group of nodes beyond those involved. However, resection was performed for ten patients (Table VII) without dissection of the submandibular lymphatics. During the dissection, periodic microscopic examination of tissue to remain has been of assistance in those cases with fibrotic tissue reaction due to radiation therapy.

The often mentioned danger of any surgical intervention about the mouth and neck following heavy doses of radiation has been circumvented by means of two factors: extending the operation beyond the anatomic limits of fibrosis and avascularity, and careful postoperative management of the surgical wound.

The early employment of urea and sulfathiazole in various concentrations, directed by a power spray into the wound, reduced the postoperative pain and swelling, the amount and severity of the infection which was invariably present, and the characteristic foul odor of oral cavity wounds. The result of daily visits to the cleanup clinic[35] in the healing of wounds after the previous employment of considerable irradiation was particularly gratifying.

TABLE VII

ONE-STAGE MANDIBULAR RESECTION AND CERVICAL LYMPHATIC NODE DISSECTION EMPLOYED IN FIFTY-SEVEN CASES OF CARCINOMA OF THE MANDIBLE

SURGICAL PROCEDURE	LOCATION OF PRIMARY CARCINOMA									
	LIP		BUCCAL MUCOSA		GINGIVA		FLOOR OF MOUTH		TOTALS	
	STAGE III	STAGE IV*	STAGE III	STAGE IV*	STAGE III	STAGE IV*	STAGE III	STAGE IV*	STAGE III	STAGE IV*
Partial mandibular resection	2	2	2	–	1	2	–	1	5	5
Partial mandibular resection and suprahyoid dissection	1	1	1	1	5	4	2	1	9	7
Partial mandibular resection and supra-omohyoid dissection	–	–	–	–	1	1	–	–	1	1
Hemisection and disarticulation	–	–	–	–	1	3	–	–	1	3
Hemisection and disarticulation and suprahyoid dissection	–	4	2	4	2	3	–	–	4	11
Hemisection and disarticulation and supra-omohyoid dissection	1	2	2	–	3	2	–	–	6	4
Totals	4	9	7	5	13	15	2	2	26	31

*Metastases confirmed by histologic examination.

Postoperatively an oral cutaneous fistula was present in twenty-nine patients. The plastic surgery procedures required for closure of the defect were performed by the plastic service after a prolonged period of observation failed to reveal any evidence of recurrence.

RESULTS

An unselected series of seventy-one cases of secondary carcinoma of the mandible is subjected to analysis. The primary lesion was in thirty-four cases controlled for variable periods of time, although only eleven patients (17 per cent) remained free from disease or died of metastases without the local recurrence of the primary lesion.

TABLE VIII

SUMMARY OF SEVENTY-ONE CASES OF SECONDARY CARCINOMA OF THE MANDIBLE

Inoperable	7	Postoperative deaths	10
Refused operation	7	Survived operation	47
Resections	57	Indeterminate (untraced)	9

	6 MO.	1 YR.	2 YR.	3 YR.	4 YR.	5 YR.	6 YR.	7 YR.	8 YR.	9 YR.	14 YR.	16 YR.	17 YR.	TOTAL
Alive, free of disease	6	4	4	2	3	–	2	1	–	1	–	1	–	24
Dead, free of disease	1	2	–	–	1	–	–	–	2	–	1	–	–	7
Alive with the disease	–	3	–	–	–	–	–	–	–	–	–	–	1*	4
Dead with the disease	2	7	3	–	–	–	–	–	–	–	–	–	–	12

Alive free of disease 3 yr. 15 (6 indeterminate) 31.9%
Alive free of disease 5 yr. 9 (8 indeterminate) 19.2%
Alive free of disease 8 yr. 6 (8 indeterminate) 12.8%

*Recurrence in skin flap raised 15 years previously from the neck to close oral cutaneous fistula.

The results of fifty-seven jaw resections undertaken for advanced carcinoma involving the mandible, and frequently associated with regional metastases, are tabulated in Table VIII. The operative mortality of fifty-seven jaw resections was 17.5 per cent.

Preliminary tracheotomy as a routine procedure has improved recent mortality figures; there were no deaths in the last group of fifteen jaw resections.

Of forty-seven patients surviving operation, 31.9 per cent lived three years; 19.3 per cent lived free of disease five years, and 12.8 per cent lived free of disease for eight years.

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JEJUNOSTOMY FOR DECOMPRESSION OF THE POSTOPERATIVE STOMACH

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DECOMPRESSION of the stomach segment following subtotal resections has been found desirable by many surgeons. The Levine or Jutte type of tube introduced through the nares preoperatively has often been left in place until secretions and food pass readily through the stoma. Some men have preferred the bidaily introduction of the nasal catheter to prevent dilatation of the stomach. Others have used intubation only to meet the need when it arises. All are agreed that convalescence is enhanced if nausea and vomiting can be avoided. Acute dilatation is a distressing complication and once it occurs, there is much delay in regaining normal tone in the stomach. The need for immediate and constant decompression has occurred so frequently in our experience that we have long used one method or another routinely. Usually, we have depended upon the inlying nasal catheter either on Wangensteen suction or simple gravity drainage. Although patients can be taught to bear with some fortitude the discomfort of the nasal tube, they all dislike it.

A few years ago, we published a report of a simple method of using a jejunostomy tube for feeding when faced with a malfunctioning stoma.[1] This technique has been used extensively in the past few years and the tube has often been placed in the jejunum at the time of the original operation. Patients in a poor nutritional state and those whose temperaments made intravenous feedings disagreeable have been benefited by jejunostomy feedings. In no instance have we met with serious complications from this procedure.

Numerous tubes have been devised to combine proximal decompression with jejunostomy feedings. Abbott and Rawson[2] advocated a double lumen tube introduced through the nares into the stomach before operation. The distal portion of the tube was introduced into the jejunum before the anastomosis was completed. This segment was used for postoperative feeding. That portion of the tube lying in the stomach segment was perforated so that suction could be applied and thus prevent dilatation.

Wangensteen[3] devised a forked catheter, one arm resting in the closed duodenal limb and one remaining in the stomach segment. He had been troubled by the temporary obstruction in the duodenal seg-

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ment and felt that decompression of this portion of the intestine was tremendously important. In our experience, the duodenal segment empties into the stomach freely enough through the anastomosis. We have been bothered, at times, by the failure of this irritating secretion to proceed into the jejunum.

Mersheimer[4] combined the Abbott-Rawson and the Wangensteen principles and devised a tube that decompressed the duodenum and the stomach and allowed the distal segment which was placed in the efferent limb of the jejunum to be used for either decompression or feeding. All of these tubes require manipulation through the partially made stoma. Although this type of contamination may often be justified, there has been considerable hesitation on the part of many surgeons to accept it.

Horsley[5] has used proximal decompression by gastrostomy in the Billroth I type of operation he prefers. A catheter is introduced through the anterior wall of the stomach and brought out through a small stab wound. This method appealed to us as ideal but is not feasible when radical subtotal gastrectomy is done, since the stomach remnant lies beneath the chest wall. This would necessitate either pulling down on the stomach segment in order to have the opening seal itself against the peritoneum when the catheter was removed, or allowing the tube to lie free for a distance between the stomach and the abdominal wall.

Bisgard[6] employed a similar gastrostomy catheter, extending it through the anastomosis into the efferent jejunal loop for feeding. Ravdin and associates[7] combined Horsley's and Bisgard's type of gastrostomy tube, using one segment for gastric decompression and the other for jejunal feedings.

In the fall of 1942, we were forced to operate for obstructing duodenal ulcer on a man who had three weeks previously been operated upon for ethmoiditis. We did not like the idea of an inlying nasal catheter and we were convinced that proximal decompression as well as early feeding would be desirable. Therefore, after a subtotal resection had been done, we introduced a No. 16 French double-eyed Bardex catheter through the jejunum and into the stomach segment. A second catheter, No. 16 French of the whistle-tip variety, was introduced a little further down in our customary manner for feeding. These were brought out through omentum and separate tiny stab wounds in the abdominal wall. Since then, we have used the proximal jejunostomy for decompression in ninety-seven patients. Many of them have had concomitant jejunostomies for feeding. In no instance has any complication arisen from these jejunostomies. Local sepsis has been inconsequential. The patients have been comfortable and no intestinal fistulas have occurred. There were four instances of lower lobe atelectasis in this group; there were no instances of pneumonia. So far, our follow-up studies revealed no complications from the jejunostomy sites.

Gravity drainage is used for three days, and then the tube is elevated to the level of the mouth. If, at the end of this time, there is a continued outpouring of secretion, further drainage is allowed. Occasionally, the tube is put on Wangensteen suction. If the amounts are

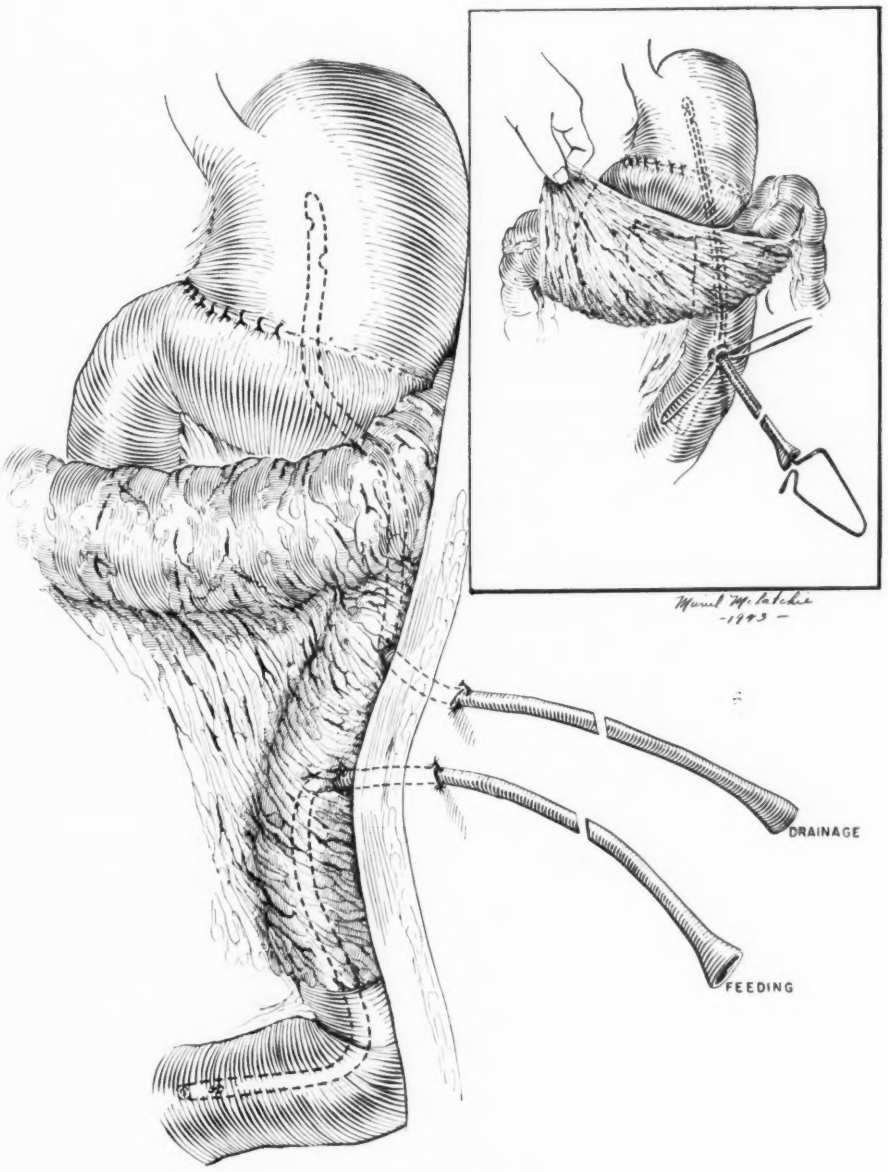

Fig. 1.

large and continue for a number of days, these secretions are added to the jejunostomy feedings. Usually the drainage is of small amount after the third day and then the tube is clamped off until it can be removed. The catgut purse-string suture deteriorates about the tenth day,

and if the tube is no longer needed, it can be withdrawn. In three instances, the catheter was not introduced far enough into the stomach segment and it dropped back into the jejunum. These patients required the use of the nasal catheter for a short period of time.

TECHNIQUE

After the anastomosis is completed, a site six to eight inches on the efferent loop of the jejunum is selected. A purse-string suture of 0 plain catgut is placed, through which the gut is perforated. A No. 16 French double-eyed Bardex catheter threaded on a lubricated stilet is then guided through the anastomosis to the center of the stomach remnant and the stilet withdrawn. The suture is tied and then the wall of the catheter is fixed at this point by the same suture, care being taken not to penetrate the lumen. A second purse-string suture of double 0 chromic catgut is then placed around the tube. A tiny stab wound is made in the left anterior axillary line well away from the laparotomy wound. A hemostat is passed through the stab wound, then through a convenient tab of omentum and the catheter is held firmly against the abdominal wall. It is fixed with a carefully placed silk suture to the skin. It is important not to penetrate the catheter at this point and not to obstruct its lumen by tying the suture too tightly.

If it is desirable to prepare for jejunostomy feedings, the second tube is placed about four inches below the first by a technique previously described.[1] A second tiny stab wound is made two inches below the first one to allow the feeding jejunostomy to be dressed separately from the drainage tube.

We have used this technique with the elimination of the inlying nasal tube following total gastrectomy for carcinoma, with equal satisfaction.

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PERFORATION OF POSTOPERATIVE JEJUNAL ULCERS

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THE problem of postoperative jejunal ulcer is one of such absorbing interest as to engage the attention of the surgeon and gastroenterologist alike. Its importance is evidenced by the fact that the frequency and severity of this complication is almost solely responsible for the current disrepute of the procedure of gastroenterostomy as a means of treatment of duodenal ulcer. That the same pathologic process may ultimately bring disrespect to the operation of subtotal gastrectomy is not beyond the realm of conjecture. In fact, recent reports make it increasingly evident that the substitution of partial or subtotal gastrectomy for simple gastroenterostomy will not, in itself, entirely eliminate this distressing consequence of gastrojejunal anastomosis. At the same time it is generally conceded that gastrectomy is less likely to be followed by jejunal ulcer than is gastroenterostomy.

By far the most dramatic, albeit the least common, complication of jejunal ulcer is free perforation into the peritoneal cavity. The first recorded case of this accident was described by Braun,[1] in 1899. Subsequently, summary reports appeared by Paterson,[2] in 1909, by Wright[3] in 1919, and Massie[4] in 1924. Ten years later, Singer and Meyer,[5] recorded 100 perforations which had occurred in 86 patients. Wilkinson[6] added two more in 1935. Bracci,[7] in 1938, found approximately 200 recorded instances of perforated jejunal ulcer in the literature. To recent reports by Lewishon,[8] Grossman,[9] Law[10] Wells,[11] Colp,[12] and Marshall and Devine,[13] totaling thirteen cases, we present four added examples of the condition, two of which occurred in the same patient. It is difficult to collect all of the recorded cases since many of them are reported as isolated case reports in communications primarily concerned with the broader aspects of peptic ulceration. Nevertheless, it may be assumed that the free perforation of a jejunal ulcer is a relatively rare intra-abdominal accident.

Singer and Meyer[5] emphasize the predisposition of patients who have experienced rupture of a jejunal ulcer to perforation in general. A surprisingly large percentage (66 per cent in one series recited by Singer and Meyer) of patients exhibiting this syndrome will have had a previous perforation of a gastroduodenal ulcer. Furthermore, the patient who experiences a perforated jejunal ulcer will frequently display a distinct proclivity toward recurrent perforation. Multiple recurrences have been reported by Robinson,[14] Brandtner and Tönnis,[15]

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Nixon and Lowry,[16] Reiss,[17] Davenport,[18] Henry,[19] and Wells.[11] One of our patients (R. F.) had a perforated duodenal ulcer, followed by two jejunal perforations, the latter two occurring only six months apart.

The symptomatology of perforated jejunal ulcer is essentially the same as that of gastroduodenal rupture, with minor exceptions. The initial pain is usually located to the left of the midline and just above the umbilicus in jejunal perforation, whereas it is higher in the epigastrium and frequently to the right of the midline in gastroduodenal rupture.

Location of these ulcers is interesting and significant. Although commonly referred to as marginal ulcer, this is a misnomer for they occur infrequently at the stoma and only rarely involve the gastric mucosa. The use of the term jejunal ulcer is therefore preferable since it is more descriptive of the pathologic process. The lesions are found entirely within the jejunum, either opposite the anastomosis or in the efferent loop, several centimeters distal to the stoma. The afferent loop is relatively immune to ulceration. When the ulceration involves the gastric mucosa, it is believed to have originated in the jejunum, and to have spread secondarily to the stomach. These areas of localization are in accord with the well-established sensitivity of jejunal mucosa to gastric secretions.

The pathology and treatment of perforated jejunal ulcers are obviously the same as those for gastroduodenal rupture.

CASE 1.—Perforation of jejunal ulcer following partial gastrectomy, operative closure with recovery, recurrent perforation of jejunal ulcer, operative closure with recovery, subsequent resection of jejunal ulcer.

R. F., (Hospital No. 217074), a white man, 37 years of age, was admitted to the Henry Ford Hospital on March 9, 1938, with a history of chronic, postprandial epigastric distress, which was associated with nausea and vomiting and relieved by milk and alkali. A definite diagnosis of active duodenal ulcer was made and medical management was instituted. The patient, a chronic continuous alcoholic with well-established psychoneurotic tendencies, failed to observe the prescribed regimen and was readmitted to the hospital for intensive therapy several times within the year. In April, 1939, he experienced a perforation of the duodenal ulcer which was closed elsewhere. Recovery, which was uneventful, was followed by an early recurrence of ulcer distress. Consequently, a partial gastrectomy was performed Nov. 1, 1940. Approximately four-fifths of the stomach was removed. A posterior gastroenterostomy was employed, the anastomosis being 20 cm. distal to the ligament of Treitz. Because of the size and acuteness of the ulcer it was deemed inadvisable to excise it, and therefore the Finsterer exclusion procedure was employed. The patient enjoyed considerable relief for several months, but later suffered a recurrence of chronic pain in the epigastrium. The suspicion of a marginal ulcer was entertained, but could not be verified by x-ray or gastroscopic examination. July 27, 1942, the patient was seized with a sudden sharp pain in the left upper quadrant of the abdomen, followed within a few hours by nausea and vomiting. On admission, the temperature, pulse, and respirations were essentially normal, the white blood count was 17,500, with 86 per cent polymorphonuclears. Abdominal examination revealed generalized tenderness, rigidity, and rebound phenomenon. A diagnosis of ruptured peptic ulcer was made and operation advised.

The abdomen was opened through an upper midline incision, under nupercaine anesthesia, revealing the unmistakable evidence of perforation of a hollow viscus. By following the streaks of fibrin and free peritoneal fluid which appeared to flow from beneath the greater omentum, a 4mm. perforation was found in the efferent loop of the jejunum about 7.0 cm. distal to the gastrojejunal anastomosis. A large indurated mass surrounded the perforation and extended into the mesentery of the jejunum. The perforation was closed with three interrupted, through-and-through, catgut sutures. A free omental graft was used to cover the perforation and the previously placed sutures were tied over the graft to secure it. One cigarette drain was inserted along the left colic gutter and another was placed into the pelvis and allowed to emerge through a suprapubic stab wound. Five grams of powdered sulfanilamide were sprinkled into the peritoneal cavity and the incision was closed with silk. The patient developed a high intestinal fistula, presumably at the site of the perforation, on the fifth postoperative day. Because of the presence of a definite hypoproteinemia, 1,000 c.c. of a 5 per cent solution of amino acids were administered daily, with rapid healing of the fistula. The patient was discharged Aug. 21, 1942.

Feb. 28, 1943, the patient was readmitted to the emergency room with the obvious signs of recurrent perforation. Through a midline incision, under spinal anesthesia, the abdomen was entered and the site of the previous perforation of the jejunum was inspected. Another perforation, apparently in the same ulcer, was found. It was repaired with a free omental graft; the abdomen was drained and closed, as before. Convalescence was devoid of untoward incident.

The patient returned to the hospital on April 28, 1943, when a lysis of the gastroenterostomy with resection of about 14 cm. of jejunum, including the ulcer, was performed. Recovery was uneventful.

CASE 2.—Perforation of duodenal ulcer, closure and gastroenterostomy; perforated jejunal ulcer; massive hemorrhage from jejunal ulcer; partial gastrectomy.

W. H. (Hospital No. 74525) was a 52-year-old white man, who had been treated for duodenal ulcer since 1926. Jan. 3, 1940, simple closure of a perforated pyloric ulcer was performed in the Henry Ford Hospital. The patient experienced postoperative pyloric stenosis which was relieved by a posterior gastroenterostomy, Feb. 29, 1940. The subsequent course was satisfactory until Nov. 26, 1942, when he was admitted to the emergency room three hours after the onset of sudden, severe, epigastric pain, which rapidly became generalized. Physical examination revealed extreme tenderness and marked rigidity of the entire abdomen. White blood count was 12,800 with 80 per cent polymorphonuclears. Flat plate of the abdomen disclosed a pneumoperitoneum.

The abdomen was opened under spinal anesthesia, revealing some free fluid and streaks of fibrin. The stomach and duodenum appeared normal. Upon reflecting the omentum and transverse colon cephalad, a 4 mm. perforation was observed in the jejunum opposite the anastomosis. A wide area of induration surrounded the perforation. The opening was closed with three through-and-through chromic catgut sutures, tied over a free omental graft. The pelvis and left pericolic gutter were drained and the incision was closed with interrupted silk sutures. Convalescence was uneventful and the patient was discharged Dec. 12, 1942.

He was readmitted March 3, 1943, with a massive hemorrhage, presumably from the jejunal ulcer. He was treated conservatively and, after a hectic course, improved sufficiently to permit his discharge from the hospital March 27, 1943.

April 23, 1943, a subtotal gastrectomy, with resection of approximately 14 cm. of jejunum, was performed. The ulcer was healed, but considerable scarring was present at the site of the previous perforation. Between three-fourths and four-fifths of the stomach was removed and a posterior gastroenterostomy was performed,

the anastomosis being approximately 19 cm. distal to the ligament of Treitz. A Hofmeister-Finsterer technique was employed. November, 1943, the patient was asymptomatic.

CASE 3.—Perforation of jejunal ulcer following gastroenterostomy.

E. S. (Hospital No. 388058), a white man, 37 years of age, was admitted to the Henry Ford Hospital, May 24, 1943. He had been treated periodically for duodenal ulcer since 1927. During the three-year interval between 1936 and 1939 he had suffered three attacks of massive hemorrhage from this ulcer. Jan. 19, 1939, an anterior gastroenterostomy with enteroenterostomy had been performed elsewhere. The patient had received considerable relief from his symptoms following the operation until the day of admission when he was suddenly seized with severe pain in the left upper quadrant of the abdomen, which came on while he was asleep, approximately three hours before admission. The pain was constant and excruciating. He had received morphine sulfate, gr. ¼, one hour before his arrival in the emergency room, but this had not relieved the pain. Examination revealed the temperature, pulse, and respirations within normal limits. The abdomen was rigid and exquisitely tender. The white blood count was 30,000, with 99 per cent polymorphonuclears. X-ray plates of the abdomen showed free air beneath the diaphragm.

The abdomen was opened through a midline incision, under spinal anesthesia. Much free fluid, fibrin, and air were present in the abdominal cavity. The stomach and duodenum appeared normal. There was a perforation, about 5 mm. in diameter, in the jejunum opposite the anastomosis. The perforation was closed with interruped chromic catgut sutures, tied over a free omental graft, after 5 Gm. of sulfanilamide had been dusted into the peritoneal cavity. The pelvis and left colic gutter were drained and the incision was closed with silk. The postoperative course was uneventful, and the patient was discharged June 7, 1943.

SUMMARY

1. Four examples of perforated jejunal ulcer are presented.

2. The term jejunal ulcer is more descriptive of the lesion than is the designation marginal or stomach ulcer.

3. Jejunal ulcers tend to occur, and to perforate, in patients who have had previous gastroduodenal perforations.

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A NEW PORTABLE SUCTION APPARATUS FOR USE WITH THE MILLER-ABBOTT TUBE

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SUCTION decompression of the intestinal tract by means of the Miller-Abbott principle has been firmly established as an important therapeutic procedure in distention due to paralytic or mechanical obstruction. Various types of suction have been devised which are satisfactory when the short gastroduodenal tube is utilized, but often considerable difficulty is encountered in securing constant suction of adequate degree when these devices are applied to a long tube. Paine,[1] in an extensive analysis of the factors operative in the use of suction for relief of distention, found a negative pressure produced by a column of water 2 feet 6 inches (56 mm. Hg) was satisfactory in most cases when a single lumen tube was used. This degree of suction is adequately delivered by the Wangensteen two-bottle apparatus. The effective negative pressure in this system is dependent upon the vertical distance from the tip of the tube in the gastrointestinal tract to the lower end of the siphon tube or the level of the water in the lower bottle if this end is submerged. Thus, the pressure will vary with the height of the bed, position of the patient, and location of the lower bottle. Abbott[2] has recently indicated that an effective negative pressure equivalent to 5 feet of water (112 mm. Hg) is required to secure adequate siphonage through the Miller-Abbott tube. This degree of suction can be produced by the three-bottle system. The variables inherent in the two-bottle system and the technical difficulties of operating the three-bottle system are minimized in the apparatus we will describe, which permits direct regulation of the degree of suction by the operator.

Our apparatus (Fig. 1) is a simple suction pump which can be cheaply assembled and will produce any desired degree of suction within safe limits. While we have seen no previous descriptions of a similar apparatus, its simplicity suggests that the principle has been used before this time. The pump can be operated by the patient (Fig. 2) in most instances—it cannot be reversed because there is only a single connection, and the degree of negative pressure in the unit does not depend on its location relative to the patient. It is a portable unit which may be carried to the bedside and suction instituted without delay. All parts liable to breakage are easily replaceable by equipment on hand in any hospital.

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The apparatus (Fig. 3) consists of a single cylinder tire pump in which the leather piston is reversed so that suction results when the plunger is pulled upward. A tire valve stem and core, from which a portion of the spring is cut away, is connected between the pump and the vacuum bottle and prevents any possibility of producing pressure in the vacuum

Fig. 1.—Apparatus attached to Miller-Abbott tube.

Fig. 2.—Patient operating suction pump.

system. The length of spring retained in the core must be just sufficient to allow easy manipulation of the plunger. The vacuum bottle is connected by tubing to a second bottle which serves as a reservoir for collection of aspirated material. An inlet tube is provided for this bottle to which the inlying tube is attached. A mercury vacuum gauge is constructed of 6 mm. glass tubing held in a 6 inch test tube by a *notched*

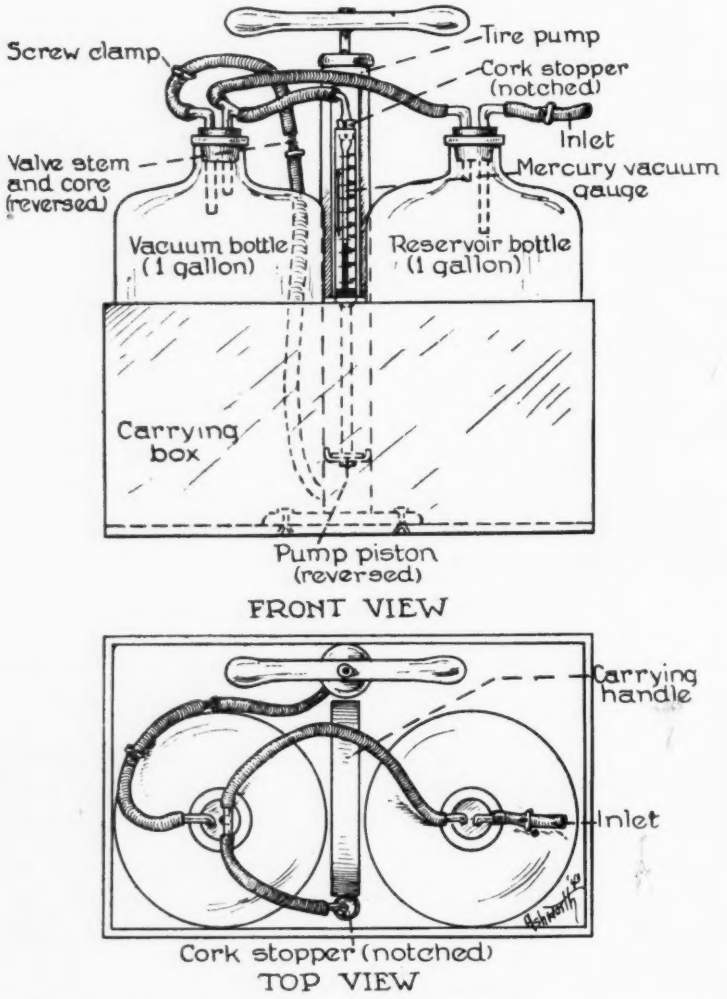

Fig. 3.—Detailed drawing of apparatus (to scale 1 inch = 6 inches).

cork to allow free communication with the outside atmosphere and is connected directly to the vacuum system. About 3 c.c. of mercury are placed in the test tube. The height of the mercury in the tubing shows the vacuum in the system at any time as measured on a millimeter scale placed behind the gauge. Marks are placed on the scale to show the desired operating range. A screw clamp is placed on the tubing between the valve and the vacuum bottle which has been found necessary because

of the tendency for the valve to leak. A second screw clamp is placed on the tubing coming to the reservoir bottle which is closed when the connection with the inlying tube is broken. In order to produce suction in the system the first screw clamp described is opened, the second one is closed, and the pump is operated. When the desired vacuum has been obtained the first clamp is closed and the second is opened to allow the suction to become effective.

During the past eight months we have utilized the apparatus in a number of cases of intestinal obstruction. In each instance the results have been gratifying to the patient, nurses, and attending physicians. Patients have remarked at the ease of operation and at no time has there been evidence of harm from the degree of suction utilized. This has been verified at autopsy by examination of the intestine. Nurses have been pleased with the simplicity of the apparatus and the infrequent attention which it requires. The only difficulties encountered were those related to leakage at the metal connector of the Miller-Abbott tube, which, however, will render any type of suction ineffective. Physicians who have utilized the apparatus have been impressed by the large quantities of fluid and gas which are removed within short periods of time and the consequent rapid passage of the tube through the small intestine.

Ordinarily when suction is effective in withdrawing quantities of fluid the plunger may have to be manipulated every two or three hours. However, after the bowel content has been largely removed, the pressure will be maintained for longer periods of time. The range of negative pressure which we have found to be most practical is between 45 and 90 mm. Hg. Between these levels approximately 500 c.c. of fluid will be removed. The presence of gas in the aspirated material will appreciably decrease this amount. In some instances it is unnecessary to have this degree of suction maintained and it is advisable to regulate this according to the amount of material coming through the tube.

Solid particles in the intestinal contents frequently cause blockage of the tube when the ordinary siphon drainage is used. Although this may occur when the present apparatus is in place, we have rarely observed any tendency for the tube to become obstructed nor has periodic irrigation of the tube been necessary.

It is possible that this type of portable apparatus may have wide applicability where low pressure suction in an airtight system is demanded.

An itemized list of materials required to assemble the apparatus which cost us $5.50 follows:

1 single cylinder tire pump
1 carrying box
2 one-gallon jugs
1 tire valve stem and core (Schrader with outside spring)
or one-way ball valve
1 three-way tube connector

1 6 inch test tube
2½ feet 6 mm. glass tubing
1½ feet rubber pressure tubing
2 No. 6 two-hole rubber stoppers
2 screw clamps
3 c.c. metallic mercury

SUMMARY

A simple, easily constructed, portable suction device is described which depends upon the fact that a tire pump will provide suction when the plunger is reversed.

The apparatus is designed primarily for use with the Miller-Abbott tube. Experiences with the suction device have demonstrated its effectiveness and simplicity of operation. Frequently the patient himself can attend almost entirely to its operation.

It is suggested that this principle may be utilized in other situations where continuous suction on an airtight compartment is desired.

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MULTIPLE PRIMARY CARCINOMAS OF THE GASTROINTESTINAL TRACT

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TO THE pathologist or surgeon very experienced in the handling of cancer material the occurrence of multiple primary carcinomas in a patient is not a rare experience. A review of the literature reveals many single case reports and series of cases. Nevertheless, the average general surgeon or internist will encounter this condition only on infrequent occasions. Such an individual, even if reasonably well informed, may not even be aware of the existence of this problem. Immediately, important questions arise. Is this a chance phenomenon? What is the correct treatment and prognosis? How great is the incidence of this lesion? Will some knowledge of the answers influence one's future approach to carcinoma cases, especially those of the gastrointestinal tract? It is with such considerations in mind that this report is submitted.

We are presenting three cases; one surgical and two found at the autopsy table. The first case also has the added interest of having presented a difficult and intriguing diagnostic problem. It stresses the nature of right colon neoplasm symptoms, emphasizing the lack of local intestinal phenomena until late in the disease, and the general character of the early symptoms.

CASE REPORTS

CASE 1.—C. C., a white woman 33 years of age, was admitted to the medical service of the Coney Island Hospital, Sept. 9, 1943. The chief complaints were marked weakness, anorexia, and continuous fever, with the temperature ranging daily from 99 to 101° F. since January, 1943.

Fifteen years prior to admission the patient had rheumatism which necessitated bed rest for one month. Two years later she noticed breathlessness on walking and occasional swelling of the ankles and legs. In the two years before admission she was subject to frequent colds and noticed occasional "heart palpitations." Six months prior to admission she was hospitalized at another institution for a month and treated for anemia. At this time there was an attack of diarrhea which lasted one week. Following her hospital discharge in January, 1943, she became progressively weaker, lost weight, and was confined to bed most of the time. In August, 1943, she had another episode of diarrhea which lasted two weeks and subsided spontaneously.

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Physical examination revealed a well-developed white woman markedly pale. The heart was enlarged to the left side. In the precordium there was a short systolic and long diastolic murmur best heard in the third left interspace. There was a short rough systolic blow at the aortic area. Abdominal findings and rectal examination were essentially negative. The working diagnosis was subacute bacterial endocarditis. Blood pressure was 164/66. Laboratory data: hemoglobin, 34 per cent; red blood cells, 2,830,000; white blood cells, 3,200; rods, 2 per cent; segmented forms, 71 per cent; lymphocytes, 27 per cent; blood platelets, 220,000. Blood culture was negative. Electrocardiogram showed marked left ventricular preponderance. Chest x-ray revealed cardiac enlargement, left ventricular. Proctoscopy was negative except for pallor of mucous membranes. Blood agglutination tests for typhoid,

Fig. 1 (Case 1).—Barium enema reveals filling defects in transverse and ascending colon. Note colon redundancy at splenic flexure area.

paratyphoid A and B, Proteus OX19, and *Brucella abortus* were negative. Stools contained no ova or parasites. Blood sugar was 107 mg. per 100 c.c. and blood urea nitrogen, 10 mg. per 100 c.c. Bone marrow examination revealed no significant variations from the normal. Urine tests were negative. Colon x-rays (Fig. 1), on Sept. 17, 1943, revealed the rectum and rectosigmoid displaced to the left. There was a partial obstruction in the proximal transverse colon with evidence of narrowing of the gut in this region. There was also a defect in the midascending colon. The cecum and terminal ileum were normal. The diagnosis was multiple tumors of the proximal colon.

The patient was then transferred to the surgical service and prepared for operation by several transfusions. Operation was performed, Sept. 27, 1943, by one of us (W. S.). A pelvic mass consisting of a right ovarian cyst was removed. Resec-

tion of the terminal ileum, cecum, ascending colon, hepatic flexure, and the transverse colon at a distance about three inches distal to the distal neoplasm with end-to-side aseptic ileotransverse colostomy was accomplished. The entire tumor-bearing area was resected together with its mesentery. There was no evidence of gross metastasis either proximate or remote.

Fig. 2 (Case 1).—Postoperative baruim enema. Note that the redundancy of the transverse colon at the left side is gone. The barium enters the small gut readily. There is a small pouch formation at the turned-in end of the transverse colon.

The patient made an uneventful recovery except for a mild wound infection. At time of discharge from the hospital three weeks postoperatively, she was afebrile and enjoyed a sense of well-being, hemoglobin and red blood cell count having risen to 75 per cent and 4,000,000 cells per c.mm. On this service we feel that the operative procedure here performed is the one of choice for neoplasms of the right colon.

Pathologist's Report.—

Gross description: The specimen was a loop of intestine consisting of a portion of ileum, ascending colon, and transverse colon measuring 45 cm. At 8 cm. from

the distal end of the transverse colon there was a cauliflower-like mass almost completely obstructing the lumen. It completely enveloped the entire circumference of the bowel and was 3 cm. in length (tumor A).

Fig. 3 (Case 1).—Gross specimen showing both carcinomas. Unfortunately, the photograph was made after fixation had caused shrinkage and distortion.

Thirteen centimeters proximal to this mass another neoplasm almost occluded the lumen of the colon. This was fungating and cauliflower-like, had a central contracted portion and some ulceration, and measured 6 by 3.5 cm. (tumor B).

Microscopic Description: In tumor A, the glands showed marked distortion and were lined by simple and multiple layers of columnar epithelium showing marked

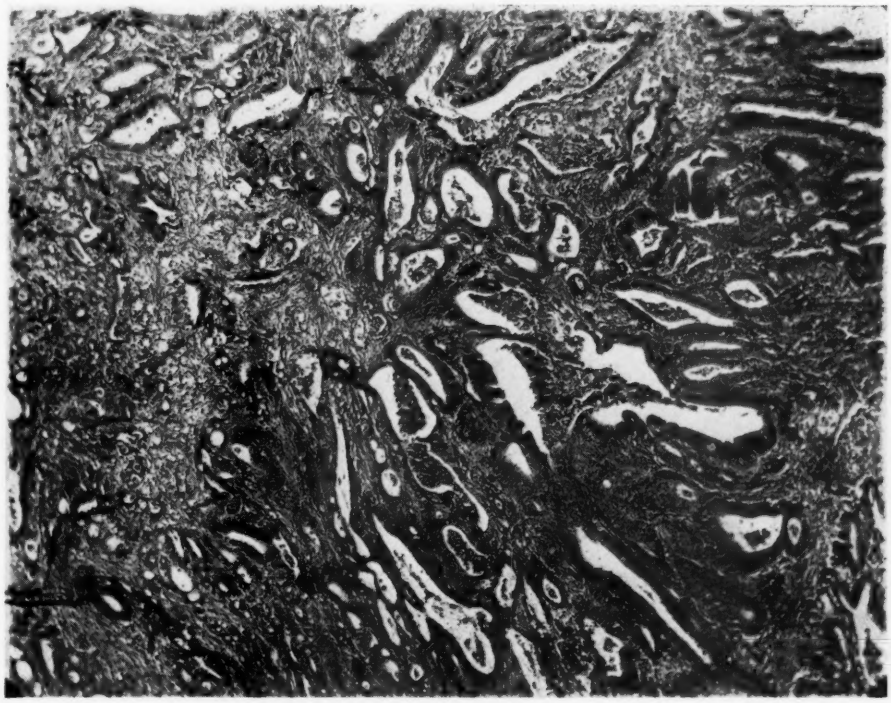

Fig. 4.—Low-power field; tumor A.

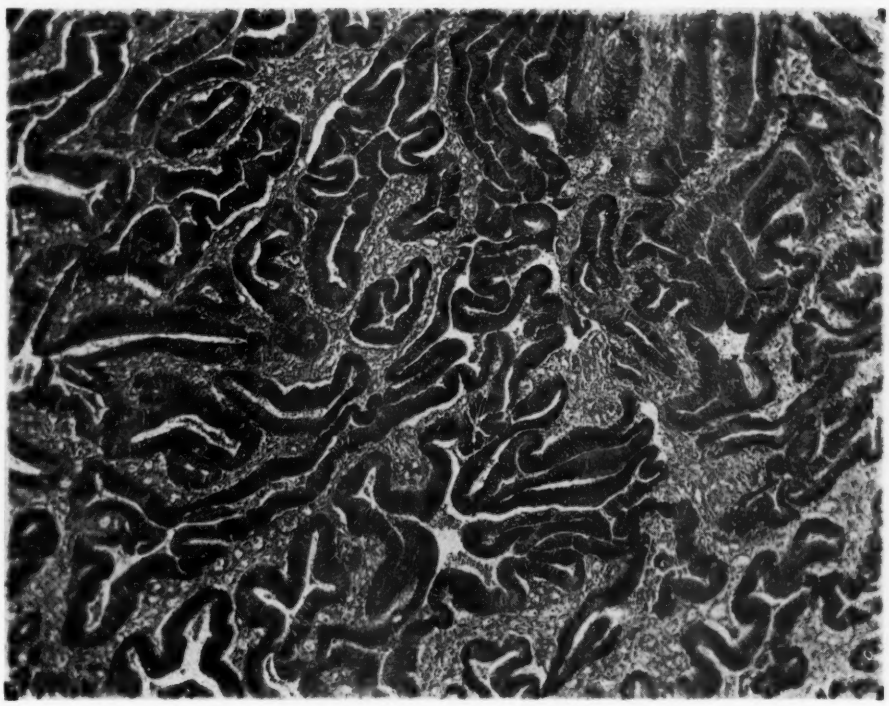

Fig. 5.—Low-power field; tumor B.

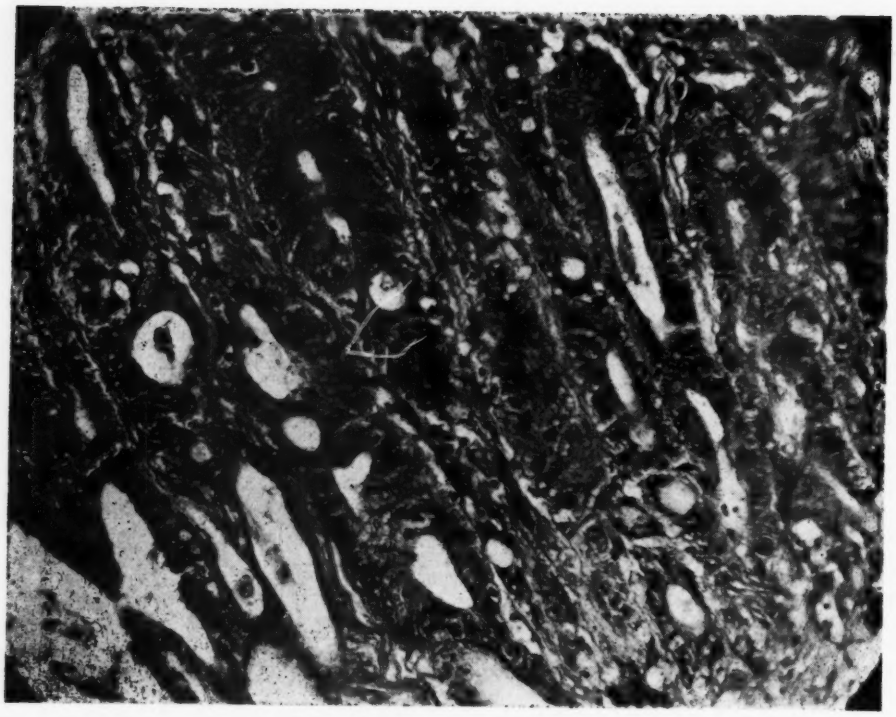

Fig. 6.—High-power field; tumor A.

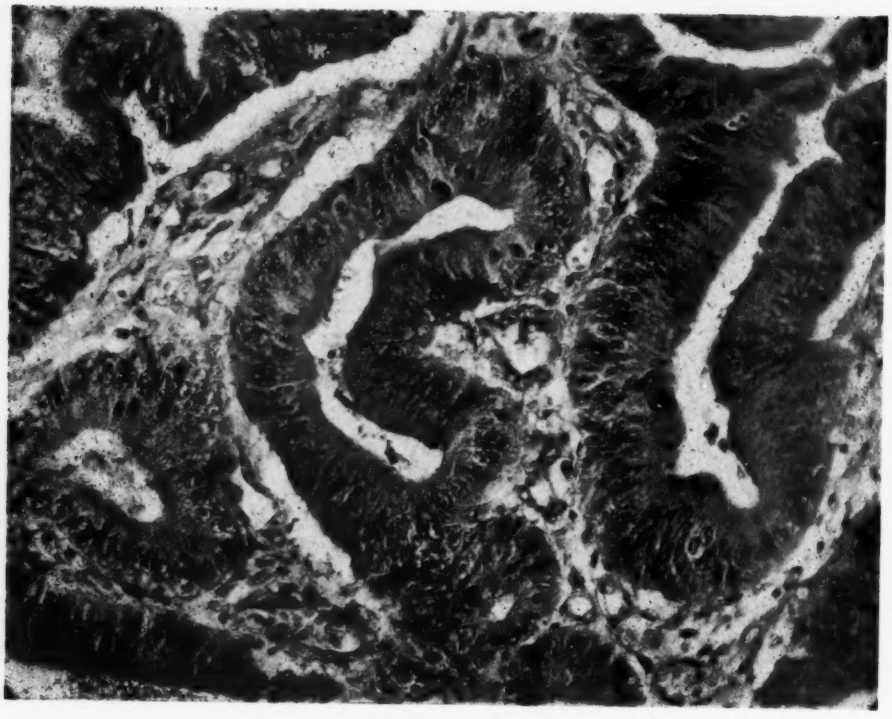

Fig. 7.—High-power field; tumor B.

variation in size, shape, and staining characteristics. There was invasion through the basal membrane into the muscularis. Many large nucleoli with hyperchromatism were noted. There was a moderate desmoblastic reaction present.

In tumor B, sections showed similar structure to that noted in tumor A, with all the criteria of malignancy present. The stroma showed a diffuse polymorphonuclear round-cell infiltration. Also noted were large focal collections of lymphocytes with tendency toward follicle formation. Note that the tumor cells of one lesion resembled those of the other but were not identical; those of the second tumor were larger. There were no evidences of metastatic relationship. There was no metastatic involvement of lymph glands. The lymph sinuses and venous channels of the intervening mucosa of the colon showed no embolic malignant cells. The cells in each case apparently arose from their individual parent mucosal cell.

CASE 2.—S. W., a white man, aged 73 years, was admitted to the Coney Island Hospital because of obstipation and distension of one week's duration. Three weeks prior to admission he noticed alternating diarrhea and constipation. The abdomen was markedly distended and rigid. There was a suggestion of an epigastric mass. While in the hospital the patient complained of substernal pain on ingestion of liquid or solid food no matter how small the amount. After two weeks he became comatose and died.

Laboratory Data.—Electrocardiogram revealed considerable myocardial damage. Blood count, urinalysis, blood sugar, and liver function tests were within normal limits. A flat plate of the abdomen was suggestive of an upper abdominal mass.

Autopsy Findings.—The liver was tremendously enlarged and was the site of widespread metastases. Numerous white nodular masses were in all the lobes. In the midportion of the esophagus there was a nodular mass 3 by 1 by 1 cm. On the lesser curvature of the stomach near the cardia there was a large ulcer 1.5 cm. in diameter. The edges were irregular and heaped. The wall was resistant to cutting. The gastrointestinal tract was otherwise negative. Microscopic examination of the esophageal mass showed diffuse squamous-cell infiltration into the muscularis with the usual criteria of malignancy. Microscopic examination of the stomach ulcer revealed the findings of adenocarcinoma. The metastatic lesions in the liver were similar to that of the stomach.

Diagnosis: (1) Squamous-cell carcinoma of esophagus; (2) adenocarcinoma of stomach with liver metastases.

CASE 3.—E. M., a white woman, aged 78 years, was admitted for hematemesis and abdominal pain of recent duration. No history of previous gastrointestinal disturbance was present. A mass about the size of a fist was palpable in the epigastrium. Pressure on this mass caused regurgitation of bright red blood. In spite of numerous transfusions and other supportive therapy, death ensued six days later. The pertinent findings at autopsy were as follows: An irregular punched out ulcer, 5 by 3 cm., was present on the posterior esophageal wall 7 cm. proximal to the cardia. The edges were sharply undermined and the base trabeculated. No regional adenopathy was present. In the prepyloric area of the stomach there was a large shallow ulcer 7 by 6 cm. on the anterior wall. The ulcer margins were heaped up. In the ulcer base an open-mouthed small vessel was present. Microscopic examination of the esophageal lesion revealed marked celluler activity with invasion of the muscularis by malignant cells showing variations in size, shape, and staining qualities. The stomach lesion had a necrotic base beneath which were confluent groups of larger cells with vesicular nuclei with prominent deeply staining basophilic nucleoli. These cells were arranged haphazardly and were found throughout all the layers of the stomach wall. No evidence of metastasis from either lesion was found.

Diagnosis: (1) Adenocarcinoma of stomach; (2) adenocarcinoma of esophagus. Each tumor was independent of the other.

DISCUSSION

In order to ascertain the incidence of multiple primary malignancies at the Coney Island Hospital, a general municipal hospital of about 300 beds, we reviewed our available figures. In the ten-year period (1934 to 1943 inclusive) 1,493 autopsies were done; of these, malignant disease was the prominent feature in 128 cases. In these 128 cases there were two cases of multiple primary malignant tumors involving the gastrointestinal tract and one involving the breast and gall bladder. In eight cases malignancy associated with a significant benign tumor in another organ was found. A review of the surgical specimens of ten years (1934 to 1943 inclusive) revealed 330 cases of malignant specimens. Of these, 110 involved the gastrointestinal tract. In one case there were two synchronous malignancies of the gastrointestinal tract. There was no history of previous malignant lesions in any of the surgical cases. Since there was no adequate provision made for follow-up study of these cases, the incidence of the future new neoplasms could not be determined.

The number of cases involved are too few for statistical study or conclusions. Nevertheless, in a ten-year period of reasonable surgical activity, only one case of multiple malignancies of the gastrointestinal tract was encountered, and in a ten-year autopsy experience, only two cases were found.

TABLE I

MULTIPLE PRIMARY NEOPLASMS OF THE GASTROINTESTINAL TRACT*

AUTHOR	YEAR	CASE
Fenger[1]	1888	1 case
Major[2]	1918	15 cases of multiple malignancies (collected from literature) in large series of multiple neoplasms (excluding mouth and pharynx)
Miller[4]	1924	5 cases
Bargen and Rankin[6]	1930	16 cases
Hanlon[7]	1931	2 cases in 18 cases of double malignancies in 710 cases of malignancy
Warren and Gates[8]	1932	59 cases (collected from literature) and 3 cases from own material (1,078 cases of malignancy)
Abel[9]	1933	2 cases from 131 cases of carcinoma of colon and rectum
Bugher[11]	1934	1 case in 30 cases of multiple malignancies in 983 malignancies
Cockkinis[13]	1934	4 cases in total of 54 carcinomas of colon and 29 cases from the literature
Brindly[14]	1938	8 cases from 306 cases of large intestinal carcinoma, 2.6 per cent incidence
Schweiger and Bargen[16]	1940	16 cases
Tullis[17]	1942	6 cases from 1,044 autopsies on malignant lesions

*Reported in the literature. (An illustrative but not complete compilation.)

Tables I and II are compilations from the literature. Table I shows the incidence of multiple primary malignancies of the gastrointestinal tract, multiple malignant polyps being excluded. Table II gives a tabulation of the general incidence of multiple primary lesions as gathered from carcinoma cases.

TABLE II

GENERAL INCIDENCE OF MULTIPLE PRIMARY NEOPLASMS*

AUTHOR	YEAR	CASE
Major[2]	1918	485 cases of multiple carcinoma (from literature)
Owen, L. J.[3]	1921	143 cases in 3,000 cases of malignancy, 4.7% incidence
Orr, J. W.[5]	1930	8 cases in 1,046 cases of malignancy (autopsy), 0.76% incidence
Hanlon[7]	1931	18 cases in 710 cases of malignancy, 2.5% incidence; 31 cases in a large group of clinical cases
Warren and Gates[8]	1932	277 cases in 20,738 cases of malignancy (European), 1.3% incidence 40 cases in 1,078 cases of malignancy (American), 3.7% incidence
Hurt and Broders[10]	1933	71 cases in 2,124 cases of malignancy, 3.3% incidence
Schreiner and Wehr[12]	1934	307 cases in 11,212 cases of malignancy, 2.7% incidence
Bugher[11]	1934	30 cases in 983 cases of malignancy, 3.1% incidence
Stalker et al.[15]	1939	113 cases in 2,500 cases of malignancy, 4.52% incidence

*Reported in the literature. (Illustrative but not complete compilation.)

Warren and Gates,[8] Hurt and Broders,[10] and Bugher,[11] after careful statistical study, conclude that the incidence of multiplicity of primary lesions is greater than chance incidence. Ewing,[18] Hanlon,[7] and Orr,[5] however, are of the opinion that multiplicity of tumors is coincidental.

Age per se was not considered the significant factor in the development of multiple lesions by Warren and Gates.[8] The age of the patients with multiple tumors was not essentially greater than those with a single neoplasm. Another factor, increased biologic susceptibility to neoplasm formation or some unknown environmental conditions favoring the same, must be considered.

The therapeutic approach in multiple primary carcinoma cases should be essentially the same as in single carcinoma cases. Care must be taken to avoid confusion with metastatic lesions. If treated promptly the outlook should be as favorable as for single carcinoma of the part involved except for the possible greater surgical risk and the theoretic increased possibility of metastasis since these may now come from more than one source.

The appearance of two synchronous independent gastrointestinal lesions will raise surgical problems depending largely on the parts involved. If the growths are in a segment of the intestine that can be treated as a unit, as in Case 1, or if both lesions are on the left side

not too far apart, the operation can be the same as for a single lesion. Radical resection with primary re-establishment of intestinal continuity for right side and transverse colon lesions, or descending and transverse colon lesions, can be readily accomplished. Low left sided lesions should be subjected to combined abdominal perineal resection and permanent colostomy. Obstructive resection will be applicable in some cases. In the event the carcinomas are widely separated, as for example a right colon and left colon lesion, then the operation can be broken down into stages, the more urgent operation being performed first to be followed at a suitable time by a second radical procedure for the other neoplasm. The problems of obstruction or other complications must be dealt with as ordinarily. Complementary cecostomy or colostomy should be used as needed. The possible combinations are of course numerous, but a radical surgical approach should be planned wherever feasible. The reappearance of a single tumor involving the gastrointestinal tract after removal of a carcinoma is not necessarily indicative of recurrence. The possibility of a second primary neoplasm must be considered and radical surgery again employed if this is the case. It is also wise in dealing with a neoplasm of the intestinal tract to exclude multiplicity, since this has an appreciable, even if small, occurrence.

SUMMARY

1. Three cases of multiple primary malignant lesions involving the gastrointestinal tract are presented.

2. Some figures illustrating the incidence of multiple primary malignancies are given.

3. The therapeutic implications are briefly mentioned.

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BILATERAL OOPHORECTOMY WITH RADICAL OPERATION FOR CANCER OF THE BREAST

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THAT the prognosis for cure of carcinoma of the breast is worse in young women when ovarian function is most active has been rather generally accepted. Thus, Ewing[1] says: "Before 30 years of age mammary cancer is extremely fatal, so that some surgeons prefer not to operate during this period. Schwartzoff reports 15 such cases, all rapidly fatal in spite of early operation." Taylor[2] writes: "There is a growing body of evidence, and rather general belief, that carcinoma of the breast in young women is more malignant and less curable than in older women." Sittenfield[3] states: "Every cancer worker realizes that cancer of the breast in a woman under 40 years of age is a highly malignant disease and notwithstanding the most thorough surgical excision and large doses of radiant energy, the end results are very grave and disappointing."

Recently, however, this opinion seems to have been modified. De Cholnoky[4] writes that in a study based on 73 cases of mammary cancer in women under 30 years of age, the results of radical surgery are comparable to those obtained in the more advanced age groups. MacDonald,[5] in an analysis of 2,636 cases of mammary carcinoma, 1,511 five-year cures, and 1,125 recurrent cases, concludes that the age of patients with mammary carcinoma does not have the prognostic significance that is commonly accredited to it. He says, however, that the best results are obtained in those patients who are 35 to 50 years of age and the least favorable in those in the decade from 50 to 60.

In an excellent report Farrow[6] of New York calls attention to certain facts that must be borne in mind when considering the effect of hormones from the gonads on the progress of malignancy of the breast or the prostate. The gonads and their products have a remarkable degree of normal variation and a multiplicity of effect. And yet by integral relationship with other organs of the endocrine system, a hormonal balance is maintained. A disturbance of this balance as caused by menopause or by castration is usually followed by compensatory changes in other endocrine organs with a partial replacement by hormones or compounds having a similar biologic effect. Thus, Woolley, Fekete, and Little[7] found a nodular hyperplasia of the adrenal cortex and subsequent proliferation of the mammary epithelium following castration of both male and female mice. Riddle[8] has reported

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that hormones capable of producing growth of the mammary tissues have been demonstrated not only in the ovary but in the pituitary, adrenal, and testes. Experimental data seem to indicate that an excess of sex hormones, whether from overactivity of the ovaries or testes or from artificial stimulation by the administration of preparations from these organs, will be similarly compensated by degeneration of other parts of the endocrine system. Such artificial stimulation is apparently a stronger influence for compensatory action than a natural overactivity.

It appears that susceptibility of response to stimulation by a specific hormone involves a hereditary factor as well as time and proper environment. Experimental and clinical data have shown that susceptibility varies in different species and in different organs, and in some instances is proportionate to the amount of hormones present. Hence, the sex hormone imbalance may be inconsistent.

Farrow says further, that in general it is believed that about one-third of the premenopausal cases of cancer of the breast can be materially benefited by castration.[9]

The beneficial influence of castration on mammary carcinoma has been observed in some cases. The fact that a few patients who have had bilateral oophorectomy for pelvic disease later developed cancer of the breast shows that the effect of castration in preventing mammary cancer is not universal, although it appears to be inhibitory.

Ewing pointed out that the cause of cancer may be classified as what he calls (1) the causal genesis, in which conditions are established that lead to the formation of cancer, and (2) the formal genesis, the biology and growth of the cancer cell itself.[10] After the causal genesis has created the conditions for the development of cancer, its function ceases and the growth of the cancer will continue of its own accord. This is well illustrated, for instance, in the carcinogenic agents, such as painting the ears of mice with coal tar or creating cancer by roentgen ray or ultraviolet light. After the cancer begins, the carcinogenic agent is unnecessary.

The relationship of the endocrines to cancer, however, seems to be of quite a different order, as Huggins has established in his experimental and clinical work on cancer of the prostate. It may well be that the hormones secreted by the ovaries act as both a causal genesis and a formal genesis of mammary cancer. Huggins and his associates have given a new light upon cancer.[11-13] Whatever may be the explanation of the benefits of orchiectomy on cancer of the prostate, and they certainly exist in many cases, it would seem that an analogy might apply to bilateral oophorectomy in cancer of the mammary gland.

Halberstaedter[14] summarized the experimental evidence of castration and of estrin on mice and rats in regard to mammary cancer as follows:

1. Early castration of female mice reduces the incidence of carcinoma in strains which show a high percentage of spontaneous mammary carcinoma.[15, 16]

2. On the other hand, the administration of estrin increases the incidence of spontaneous carcinoma in female mice in strains which are characterized by a low percentage of spontaneous mammary tumor.[17]

3. Administration of estrin to male mice of such strains produces mammary carcinoma, though otherwise they never develop tumors.[18, 19]

4. Mammary carcinoma may be produced in male mice by implantation of ovaries following previous castration.[20]

5. Administration of estrin produces mammary carcinoma in rats belonging to a strain in which no spontaneous mammary tumors occur.[21]

6. The time required for the appearance of the mammary cancer was reduced if a higher daily dose was injected or if compounds of greater estrogenic potency were administered.[21]

While the causal relationship of excessive estrogenic substance to mammary cancer in mice and rats is quite clearly demonstrated, clinically this relationship is difficult to prove absolutely. However, cases in which it seems probable have been reported.[22-26] Hamblen states: "Untoward responses to estrogenic therapy are increasing since the availability of cheap, orally active estrogens of the diethylstilbestrol type; these are due chiefly to overdosage. Diligence to avoid overtreatment should be observed." It appears proved, however, both experimentally in mice and rats and clinically, that a family history of mammary carcinoma has much to do with the incidence of this disease.

That some cases of well-developed mammary cancer have been benefited by removing both ovaries is fairly well established. There are instances of apparently complete relief.[27, 28]

What seems to be the double role of estrin in the causal and in the formal genesis of mammary cancer resembles what might be the relationship between androgenic substances from the testicle and cancer of the prostate. The beneficial results of removing the ovaries along with a radical operation for cancer of the breast may be attributed to the withdrawal of a causal genesis, to the removal of a formal genesis, or to a combination of both of these factors. The recurrences of cancer of the breast after a radical operation are doubtless due to cancer cells that have been left. The small amount that remains, however, would be stimulated by estrogenic substances. As there have been cases in which cancer of the breast receded, at least temporarily, after removal of the ovaries with no other treatment, it would seem that there may be a mass relationship. That is, the effect of withdrawing

estrogenic stimuli might be greater if there are only a few cancer cells than if there is a large amount. This may be roughly compared, in reverse, with the action of the antitoxin of diphtheria, which must be given in at least a certain minimum quantity in order to obtain a desirable therapeutic result. When cancer is far advanced, it may be that changes occur in the metabolism of the cancer that will permit its growth and the cancer increases, even though the promoting influence from estrin is no longer present. With only a few remaining cancer cells, however, this unfavorable influence of withdrawing the stimulating effect of estrin should be more deleterious and create an unfavorable soil for their existence.

Complete abolition of all ovarian function cannot always be obtained except by bilateral oophorectomy. Huggins states that the interstitial cells of the testicle are not usually destroyed by irradiation.[11] The ovaries are much more protected from the effects of irradiation than are the testicles. Kaplan[29] reports twin pregnancy after temporary suppression of menstruation following roentgen-ray treatment for mammary cancer. This shows that sterilization by irradiation of the ovaries is not always effective. Some patient will doubtless react differently and require a larger dose of irradiation than others.

In women who have previously had cancer in one breast, the increase of ovarian activity in pregnancy appears to promote mammary cancer in the remaining breast. Trout[30] collected fifteen instances of pregnancy subsequent to a radical operation for mammary cancer, in thirteen of which there was prompt development of very malignant carcinoma in the remaining breast. Wintz[31] reported seven instances of pregnancy after radical operation for mammary cancer, with fatal cancer then occurring in the remaining breast.

My experience with the ultimate results of radical operation for cancer of the breast in young women has been rather disappointing (Table I). Of nine patients under 36 years of age, on whom radical operation

TABLE I

OPERATIONS FOR CARCINOMA OF THE BREAST IN PATIENTS UNDER 36 YEARS OF AGE BY J. SHELTON HORSLEY AT ST. ELIZABETH'S HOSPITAL (SEPT. 1, 1922, TO NOV. 1, 1937)

	DATE	OPERATION	LOCAL X-RAY	AGE	BREAST	AXILLARY NODES	GRADE	RESULT
1.	9/13/22	Rodman	0	34	L.	x	4	Died, metastasis to spine
2.	7/ 8/25	Rodman	x	34	R.	0	2+	Died, metastasis to lungs
3.	8/ 5/25	Rodman	0	33	L.	0	3	Died, metastases to lungs and supraclavicular space
4.	9/ 8/28	Rodman	x	34	R.	x	4	Died, metastases to brain and lung
5.	3/19/31	Rodman	0	33	R.	0	2	Alive and well
6.	4/ 7/31	Rodman	0	33	R.	0	1+	Alive and well
7.	5/12/35	Rodman	x	34	R.	0	3	Died, metastases to bones
8.	4/16/36	Rodman	x	28	R.	0	4	Alive and well
9.	10/ 4/37	Rodman	x	35	L.	x	3+	Alive and well

Nine cases, 5 died with metastatic cancer; no local recurrences. (Note Case 6 was grade 1 with no axillary nodes involved.)

was done, from Sept. 1, 1922 to Nov. 1, 1937, five have had recurrence (55.5 per cent). In one of the cases without recurrence the cancer was grade 1 with no involvement of lymph nodes, a condition very favorable for cure. Of the total cases of radical operation (148, including these 9), for this same period, there was one operative death from septicemia. There were seventy-five recurrences (51.02 per cent). The results of experimental work on mice and rats which has been referred to, and the clinical results obtained by Beatson, Lett, Torek and others, showing occasionally some benefit in advanced mammary cancer after bilateral oophorectomy, caused me to adopt the procedure of removing both ovaries whenever a radical operation was done for cancer of the

TABLE II

RADICAL OPERATIONS FOR CARCINOMA OF THE BREAST WITH BILATERAL OOPHORECTOMY BY J. SHELTON HORSLEY AT ST. ELIZABETH'S HOSPITAL
(NOV. 1, 1937, TO OCT. 1, 1943)

	DATE	OPERATION	AGE	BREAST	AXILLARY NODES	GRADE	RESULT
1.	11/19/37	Rodman	33	L.	x	4	Alive and well
2.	11/10/39	Rodman	38	R.	0	3+	Alive and well
3.	8/10/40	Rodman	42	L.	0	3+	Alive and well
4.	2/ 5/41	Rodman	38	L.	0	3	Alive and well
5.	2/14/41	Rodman	40	L.	x	2	Alive and well
6.	4/25/41	Rodman	47	R.	0	2	Alive and well
7.	4/26/41	Rodman	31	R.	x	3	Alive and well
8.	5/30/41	Rodman, bilat.	28	R. L.	x x	3+ 3	Died 6 months after operation; metastases to lung and skin
9.	6/17/41	Rodman	38	L.	0	3	Alive and well
10.	9/ 1/41	Rodman	33	L.	x	4	Alive and well
11.	10/15/41	Rodman	40	R.	0	2	Alive and well
12.	1/13/42	Rodman	39	R.	x	3	Alive and well
13.	2/16/42	Rodman	47	L.	x	3	Alive and well
14.	3/21/42	Rodman	44	L.	x	3	Alive and well
15.	4/13/42	Rodman	43	R.	0	3	Alive and well
16.	6/15/42	Rodman	43	R.	0	2 mucoid	Alive with metastases
17.	7/ 4/42	Rodman	28	R.	0	3	Alive and well
18.	9/25/42	Rodman	35	L.	0	3	Alive and well
19.	10/ 5/42	Rodman	39	R.	0	3	Alive and well
20.	11/23/42	Rodman	30	R.	0	3	Alive and well
21.	2/23/43	Rodman	45	L.	0	3	Alive and well
22.	3/30/43	Rodman	44	R.	x	3	Alive and well
23.	7/ 8/43	Rodman	33	L.	x	3	Alive and well
24.	8/18/43	Rodman	37	L.	0	3	Alive and well
25.	9/22/43	Rodman	41	R.	0	2	Alive and well

NOTE.—It is, of course, not claimed that any of these patients are "cured" in the sense of an impossibility of a return of the cancer. It is well known that there are occasional cases of recurrence of cancer of the breast after much longer periods after operation than five years. All of these patients either have been examined by me or by some of my associates, or have been communicated with during the latter part of 1943 or later. The first patient (No. 1, Table II), who had a recurrence from a partial operation done elsewhere, was operated upon by me Nov. 19, 1937, more than six years and one month before submission of this article for publication. In my own experience the majority of recurrences from cancer of the breast occur within eighteen months after operation. It will be noted that the results in those cases without recurrence are classified not as "cured" but as "alive and well." In the last six cases in Table II the operations have been too recent to have significance as far as "so-called cure" is concerned, but in nineteen of the cases the results reported are for periods varying from fourteen months to about six years and one month after operation, with only two recurrences. All cases have been traced.

breast in young women. About four years after my first operation of this kind, which was on Nov. 19, 1937, the splendid report of Huggins on castration for cancer of the prostate appeared. His work seems to confirm the idea of bilateral oophorectomy in premenopausal women whenever a radical operation is done for cancer of the breast. This procedure apparently was first suggested by Schinzinger,[32] although according to his article, he did not carry it out. He is said to have discussed this also before the Surgical Congress in Berlin on April 25, 1899. Doubtless it has been performed by other surgeons, although there does not seem to be any systematic record of a number of cases.

At first I did bilateral oophorectomy with radical operation for cancer of the breast only on patients under 40 years of age. Later I extended it to all patients in the premenopausal stage. The desirability of having both ovaries removed along with the radical operation is, of course, first explained to the patient. At present I adopt the position that if the patient is unwilling to have this done, I will not treat her. Since extending this operation to all premenopausal patients there has been an increase in the number of these cases.

From Nov. 1, 1937 to Oct. 1, 1943, I have performed this operation of bilateral oophorectomy with the radical operation for mammary carcinoma on twenty-five patients, the first case on Nov. 19, 1937, the last on Sept. 22, 1943. There have been only two recurrences, one in a patient with bilateral mammary cancer who doubtless had internal metastases at the time of operation, although they could not be demonstrated then, and the other in a patient with mucoid cancer. Fourteen of these patients were operated upon since Jan. 1, 1942 (Table II).

A report of three of the earlier cases with previous incomplete operation or biopsy done elsewhere is of interest:

CASE 1 (No. 1 in Table II).—Mrs. C. B. A., age 33 years, had noticed a small lump in the left breast in January, 1937. Her mother died of nephritis and carcinoma of the left eye. On June 11, 1937, a left local mammectomy had been done elsewhere. Microscopic examination showed cancer. She then had thirty roentgen-ray treatments over the region of the left breast and axilla. Six weeks after these treatments she noticed a small lump in the left axilla and was given fifteen more roentgen-ray treatments, but the lump remained. Examination on admission to St. Elizabeth's Hospital, Nov. 18, 1937, showed the skin over the left thorax and left axilla brown from the roentgen-ray treatments and there were palpable lymph nodes in the left axilla. On Nov. 19, 1937, a radical operation for recurrent carcinoma of the left mammary gland and bilateral oophorectomy were performed. This was the first case in which this combined operation was performed. Microscopic examination of the axillary lymph nodes showed adenocarcinoma, grade 4. In some cells there was evidence of the effect of irradiation, but in most areas the cancer cells did not seem to be affected (Fig. 1). The patient was well, with no recurrence, on Feb. 10, 1944, more than six years and two months after operation. She appeared to be in perfect health.

CASE 2 (No. 3 in Table II).—Mrs. R. P. H., age 42 years, was admitted to St. Elizabeth's Hospital, Aug. 10, 1940. Three weeks before admission the patient

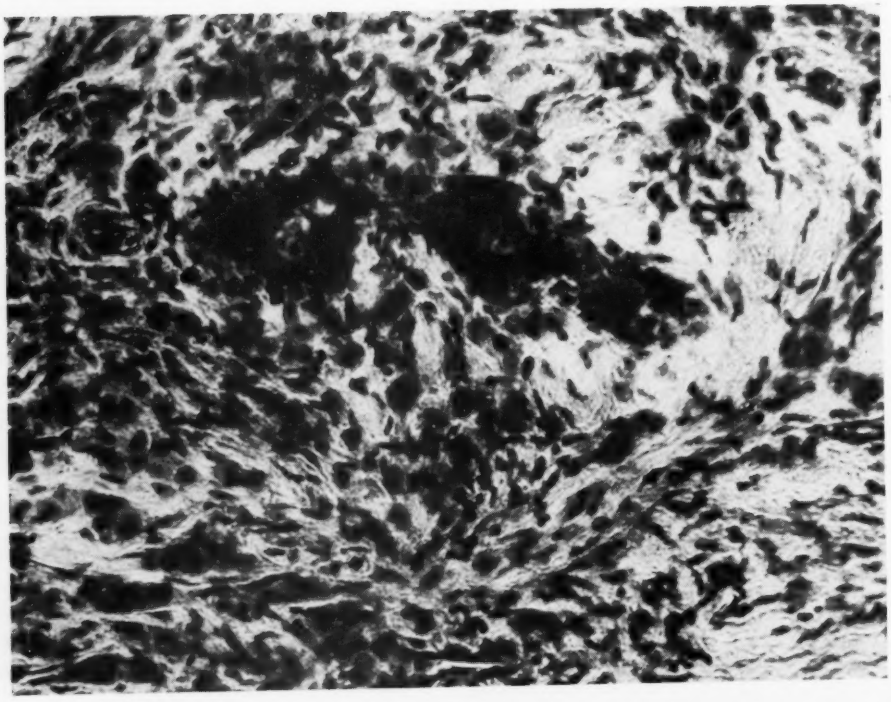

Fig. 2.—Case 2, Mrs. R. P. H., adenocarcinoma, grade 3 plus (×240).

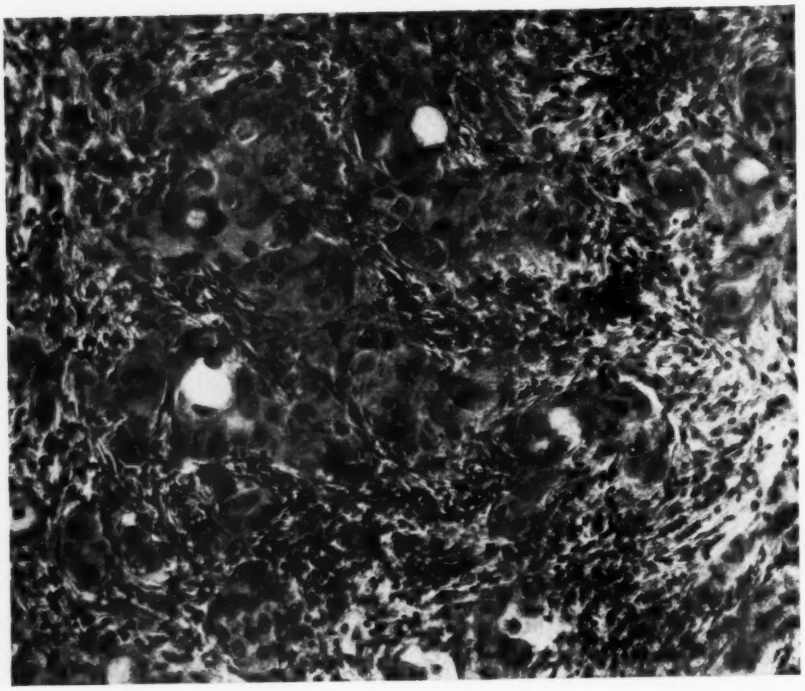

Fig. 1.—Case 1, Mrs. C. B. A., adenocarcinoma, grade 4 (×180).

had noticed a small, hard, painless mass in the upper outer quadrant of the left mammary gland. One week before admission the tumor had been removed elsewhere and microscopic examination showed cancer. She was otherwise in good condition. On Aug. 10, 1940, a radical operation for carcinoma of the left mammary gland and bilateral oophorectomy were performed. Microscopic examination showed adenocarcinoma, grade 3 plus (Fig. 2). The lymph nodes in the axilla were hyperplastic, but apparently not cancerous. This patient was in good health and had no recurrence when examined on Nov. 16, 1943, three years and three months after operation.

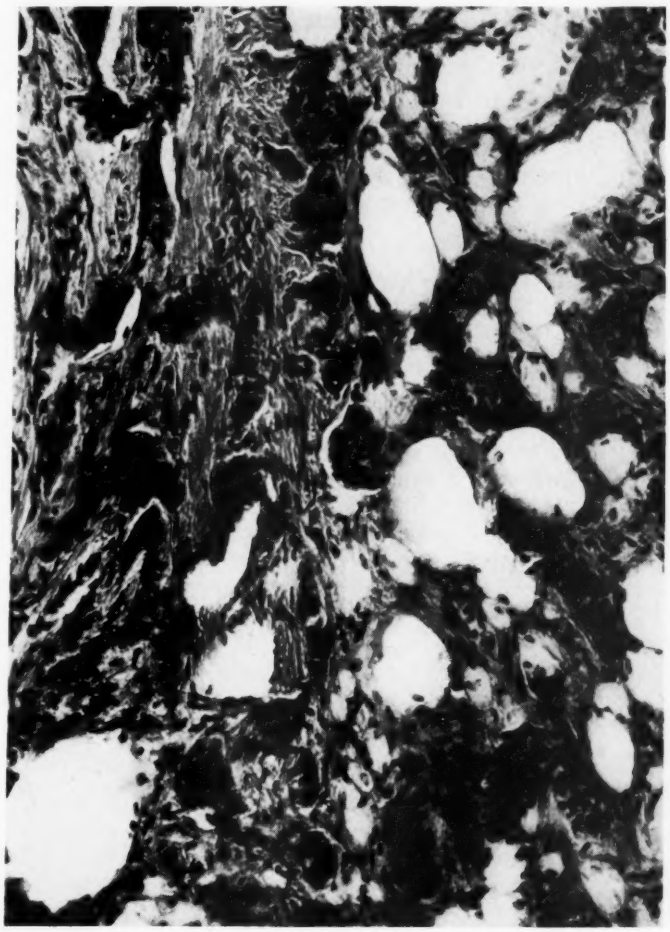

Fig. 3.—Case 3, Mrs. A. D. W., adenocarcinoma, grade 3 (×180).

CASE 3 (No. 4 in Table II).—Mrs. A. D. W., age 38 years, was admitted to St. Elizabeth's Hospital on Feb. 3, 1941. Her mother died of cancer of the ovary. In June, 1940, a small tumor was removed from the patient's left breast elsewhere. Two months before admission to St. Elizabeth's Hospital she noticed another small tumor just lateral to the old incision. There had been infrequent sharp pains. On admission there was a scar in the upper outer quadrant of the left breast and near it were four firm, round tumors. There were no palpable lymph nodes in the axilla. Feb. 5, 1941, a radical operation for carcinoma of the left mammary gland

was performed with bilateral oophorectomy. Microscopic examination showed cancer, grade 3 (Fig. 3). When examined on Dec. 13, 1943, two years and ten months after operation, there had been no recurrence.

Another patient in this series is quite interesting:

CASE 4 (No. 7 in Table II).—Miss F. A., aged 25 years, was operated upon by me April 2, 1935, a radical operation being performed for cancer of the left mammary gland. Removal of a small tumor which was carcinomatous had been done elsewhere March 22, 1935. She married (now Mrs. G.) and had a baby. On April 23, 1941, a small tumor of the right mammary gland was removed elsewhere, with a histologic diagnosis of cancer. April 26, 1941, I performed a radical operation for cancer of the right mammary gland and bilateral oophorectomy. The baby was then about 18 months old. The patient was last examined on Nov. 1, 1943 and there was no evidence of recurrence.

It is well known that patients with cancer of the breast who have been previously operated upon by local removal of a cancerous tumor or by biopsy, with an interval of several days or longer before the radical operation, are much more likely to have a recurrence than those on whom a radical operation has been the primary procedure. All four of these cases are in this class. Case 4 also illustrates the observation made by Trout and others, that after removing one breast for cancer, pregnancy is prone to initiate cancer in the other breast.

CASE 5 (Mucoid cancer, No. 16 in Table II).—Mrs. F. H. S. had a radical operation for cancer of the right mammary gland and excision of both ovaries on June 15, 1942. Microscopic examination showed mucoid cancer (Figs. 4 and 5). The lymph nodes in the right axilla were enlarged, but microscopic examination showed only hyperplasia and no metastasis. This is the only case in the series of twenty-five in which there was mucoid cancer. The patient had arthritis involving the lumbar spine, which was revealed by roentgen-ray examination on Jan. 8, 1943. She continued to suffer indefinite pain, although there was then no evidence of local recurrence or of metastasis in the thorax or in the neck. Examination on Dec. 10, 1943 showed the scar in good condition and no evidence of recurrence, although there was much discomfort and pain, chiefly around the right hip and spine. Roentgen-ray examination, Jan. 31, 1944, revealed an erosion of the left eighth rib and the right seventh rib in the posterior axillary plane. There was also an absence of the spinous process of the fourth thoracic vertebra. These findings indicate metastases.

Ewing[33] states that the mucinous material in this type of cancer is usually in the stroma with the epithelium compressed in small islands of inactive cells. The mucoid substance is generally outside of and surrounding a group of compact, well-preserved cells. This is not in accordance with the epithelial origin of the mucinous material and has not been satisfactorily explained by those who maintain this origin. Ewing states, "In four cases I have not seen mucous degeneration of epithelium, but the compact alveoli lay in broad areas of mucinous stroma. There was strong evidence that the mucinous change had involved and partly originated in the fat tissue in the stroma."

The mucoid type of cancer of the breast is uncommon. It grows more slowly and apparently is less malignant than the usual type of

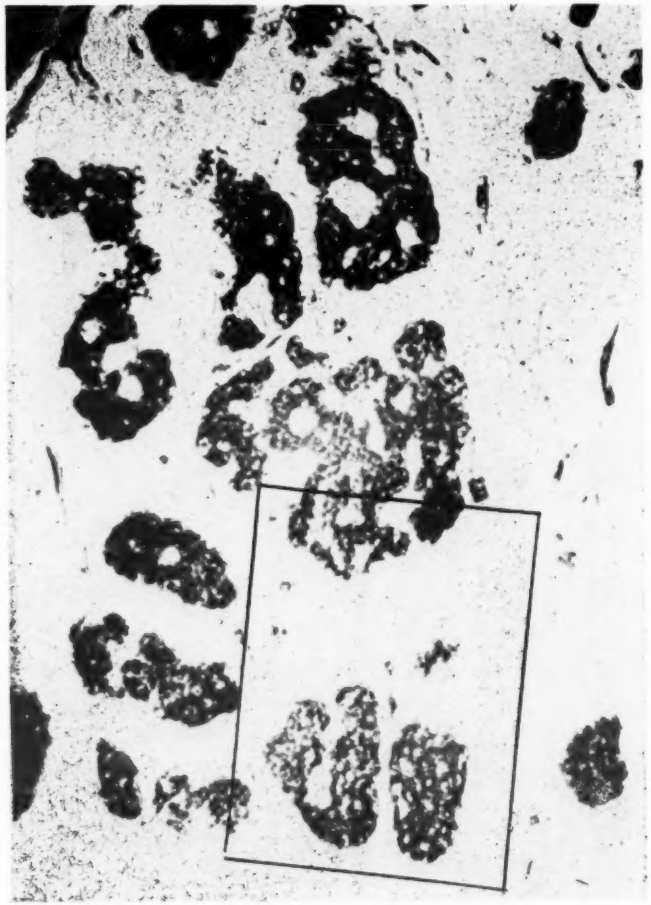

Fig. 4.—Case 5, Mrs. F. H. S., mucoid cancer of the breast showing islands of epithelial cells floating in a mucinous material (×108).

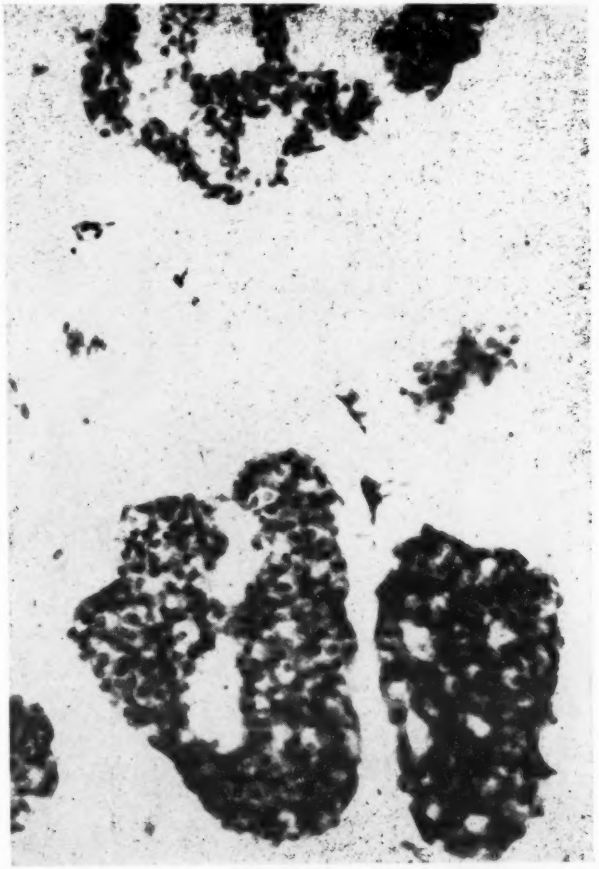

Fig. 5.—Case 5, Mrs. F. H. S., higher power from lower portion of Fig. 4 (marked off) (×240). The epithelial cells do not appear to be active. It seems probable, as Ewing says, that the mucinous material originated from the stroma or from the fat in the stroma.

mammary cancer. It tends to metastasize later. It would seem then that the pathogenesis of mucoid cancer of the breast differs from that of the usual mammary carcinoma, and it may well be that it is not influenced by estrin products, as it probably arises from the stroma. This is further emphasized by the fact that the epithelium in the islands in the mucinous substance is not active.

Therefore, it appears unnecessary to remove the ovaries in a case of mucoid cancer of the breast. The neoplasm should be examined immediately after the radical operation on the breast and before attempting a bilateral oophorectomy which seems to be efficient in preventing recurrence after a radical operation for the usual type of mammary cancer, but will doubtless be of no avail in mucoid cancer of the breast.

SUMMARY

The reasons for adopting bilateral oophorectomy along with the radical operation for mammary cancer in premenopausal women are discussed. The results of twenty-five cases so treated are presented.

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INTERNAL FIXATION OF FRACTURES OF THE PATELLA WITH COTTON SUTURE MATERIAL

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VARIOUS suture materials have been used for internal fixation of fractures of the patella including iron alloy wire, silver wire, silk, linen, catgut, and kangaroo tendon. The multiplicity of materials used at once suggests the inadequacy of any one of them.

In 1939, Meade and Ochsner[2] emphasized the advantages of cotton suture material experimentally and clinically in general surgery and it is now the consensus of opinion that cotton as compared with other materials produces the least amount of tissue reaction and yet maintains its tensile strength.

Although today wire is the most popular suture material for repair of fractures of the patella, experience has shown that cotton has many superior qualities: (1) Undesirable electrolytic reactions which are associated with wire do not occur with cotton. Electrolysis causes disintegration of metal which results in increased tissue reaction and weakening of wire.[5] (2) Cotton may be tied rather than twisted and thus breaking either at the time of operation or later is not a problem. (3) Joint reactions often associated with the presence of wire[6] have not been observed with cotton.

Other noteworthy attributes of cotton have demonstrated its advantages over silk, linen, kangaroo tendon, and catgut, which are less commonly used than wire in repairing patellar fractures: (1) Cotton can be easily and thoroughly sterilized by boiling for twenty minutes or autoclaving for ten minutes at fifteen pounds' pressure.[4] Of course, in boiling, shrinkage of the material must be considered and the cotton should not be wound on a rigid spool. (2) Cotton maintains its tensile strength 100 per cent at the tenth day of wound healing.[3] (3) It causes little tissue reaction.[3] (4) A practical consideration is the availability of mercerized or nonmercerized cotton at any dime store at nominal cost.

In this study, cotton suture material was used for internal fixation of simple transverse fractures of the patella having wide separation of fragments. In this type of fracture it is usually possible to secure *an exact reduction with a smooth articular surface* by operation.

TECHNIQUE

Following a forty-eight-hour skin preparation, under general anesthesia, a slightly curved transverse incision is made from medial to

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lateral femoral condyle, the apex of the incision being slightly distal to the distal fragment (Fig. 1*a*). Bleeding points are ligated with fine cotton (No. 80), skin draping is affixed, fragments are exposed, and interposing soft parts removed. The knee joint is thoroughly irrigated with warm normal saline solution and old clots removed. With a seven-sixty-fourths-inch drill a transverse hole is made in each fragment about one-half inch from the fracture line (Fig. 1*b* and *c*) and a double strand of crochet cotton (No. 10), which has been previously wet and tested by the operator to show up possible defects in the suture, is threaded on a Keith needle or probe, passed through the holes, and tied firmly with a triple knot while the assistant holds the fragments in apposition. Two accessory anterior sutures of No. 10 cotton are then placed after ap-

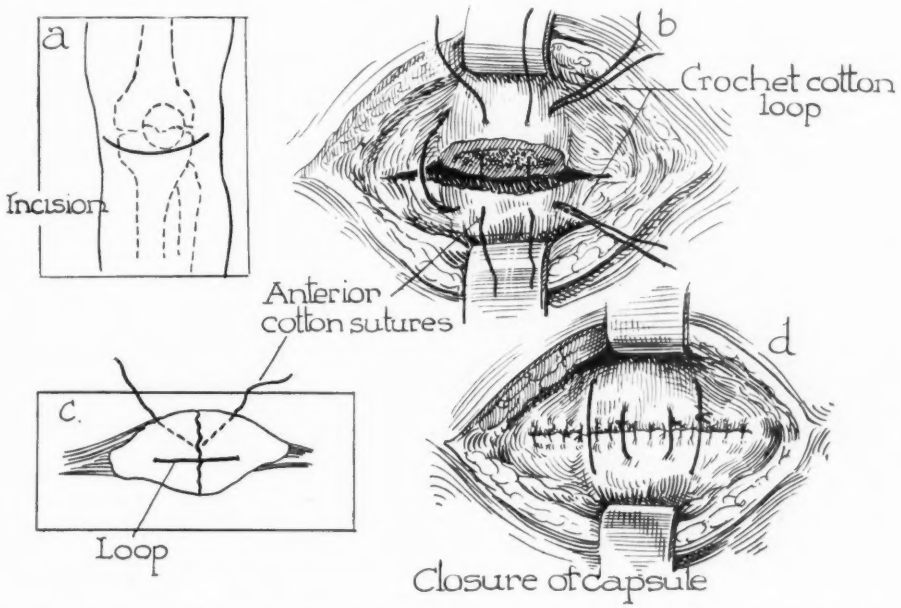

Fig. 1.—(*a*) Slightly curved transverse skin incision, (*b*) double strand No. 10 crochet cotton loop and two anterior sutures of crochet cotton, (*c*) sagittal section showing loop and anterior suture through patella. (*d*) Patella is repaired with crochet cotton and capsule closed with interrupted sutures of quilting cotton.

propriate holes have been drilled (Fig. 1*b* and *c*). Drill holes must never penetrate or undermine articular cartilage. The knee is flexed slightly to determine whether the fracture is securely fixed and the articular surface of the patella is checked for anatomic reduction. The articular capsule and the quadriceps expansion are then repaired medial and lateral to the patella with interrupted sutures of quilting cotton. These sutures are all inserted before any one of them is tied in order to create equal tension on all sutures, and are closely spaced to obtain hemostasis in the synovial capsule (Fig. 1*d*). The subcutaneous tissues and skin are approximated with interrupted sutures of quilting cotton. The wound is dressed, a posterior plaster splint is applied from the ischial

tuberosity to beyond the toes with the knee in extension, and the hip is flexed to 135 degrees for two days to relax the rectus femoris muscle. After three weeks the splint is removed and active motion, heat, and massage are started.

A midthigh tourniquet is not used because it frequently interferes with passive stretching of the quadriceps group, especially the rectus femoris, and thereby makes apposition of fragments more difficult.

A. *B.*

Fig. 2.—*A*, Transverse fracture showing patella with separation of fragments. *B*, Same patient seven weeks following repair of patella with cotton; this patient had a complete painless range of motion at this time.

DISCUSSION

Eleven patients with patellar fracture have been treated by this method and a similar procedure has been employed in seven cases of transverse intra-articular fracture of the olecranon process. The crochet cotton loop and a similar technique have also been used by Dixon[1] in six cases of patellar fracture. In none of these twenty-four cases has there been any wound infection and in no case has removal of the cotton loop been necessary. All of the patients had a useful range of motion within six to eight weeks and were able to resume their regular occupations at that time (Fig. 2*A* and *B*). As might be anticipated, there is minimal joint effusion and minimal local reaction to the cotton loop and cotton sutures.

Of minor consideration is the fact that cotton, unlike wire, does not appear in the x-ray film and with certain patients this has a psychologic advantage.

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EXPERIMENTAL HEAD INJURY PRODUCED BY BLASTING CAPS

An Experimental Study

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RECENT studies indicate that head traumas vary in character with the type and amount of violence applied. During the present war it has been observed repeatedly that patients with penetrating wounds of the head or localized depressed fractures were not unconscious.[1, 2] The studies of Denny-Brown and Russell[3] have shown that concussion, as they defined the word, resulted from a blow upon a large surface of the skull which, as a rule, produced no fracture. They also showed that the strength of the blow was an important factor in producing concussion; they measured this amount of violence, and expressed it in terms of "head acceleration" (28 feet per second).[3]

It is clear, therefore, that the approach to the problem of the closed head injury must be made under well-defined standardized conditions in the laboratory. Previous experimental work on head injury is open to criticism because of the techniques employed in producing the trauma. As Denny-Brown and Russell[3] pointed out, most investigators have fixed the head before delivery of the blow. Frequently, multiple hammer blows of unequal and unknown intensities producing localized depressed fractures have been used.[4-6] Other workers have employed mechanical devices, such as falling objects and pendulums, and left it uncertain if any serious injury was produced after a single blow.[7, 8]

To produce a severe type of standardized head trauma without fracturing the skull at the site of impact, it was felt that the following criteria should be satisfied:

1. The same amount of violence should be employed in all experiments.
2. The skull should remain intact at the site of impact.
3. The animal should be severely disabled as a result of the single blow.
4. The head should be freely movable.
5. The trauma should be confined to the head only.

In our experience with the adult dog, these criteria cannot be satisfied by the usual mechanical devices used to produce experimental head

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injury. Following single blows with the pendulum or falling objects, even when concussion occurs, the dogs show little disability after an hour and seem to recover completely. Manually delivered blows with a baseball bat or mallet at times produce a seriously disabled dog, but possess the obvious disadvantage of not being uniform. We found that the criteria mentioned here could be fulfilled by using a standard commercial blasting cap which could be applied to any chosen surface of the head. The explosive used in this study inflicted violence which was well localized to the head, and in no way produced any injury to the lungs or other major organs. The purpose of this communication is to report the experimental technique used to produce head injury, the accompanying neurologic findings, and the gross pathology. The histopathology of the brains will be reported in a later article.

Fig. 1.—Du Pont No. 6 electric blasting cap.

The explosive selected was the du Pont No. 6 Electric Blasting Cap. Its chief commercial function is detonation, and it is used to fire high explosives. Mr. F. F. Chapman of the explosives department of the E. I. du Pont de Nemours & Company was kind enough to furnish the following information and the accompanying sketches (Figs. 1 and 2).

"The standard du Pont No. 6 Electric Blasting Cap has a 1⅝ inch long copper shell containing a base load of tetryl, a pressed primer load,

and a loose ignition charge. The electrical firing element consists of two leg wires, a sulfur composition bridge plug which holds the two leg wires in place, and a small diameter corrosion-resistant bridge wire attached across the terminals of the leg wires about $\frac{3}{16}$ inch below the plug. This firing element is placed in the cap so that the bridge wire is embedded in the loose ignition charge, and then sealed in place by a pour of water proofing material and finally a pour of sulfur. Upon application of electrical current the bridge wire heats up to incandescence and fires the cap. The detonation speed of tetryl, the base charge material in the cap, is approximately 17,000 feet per second. We are unable to give any information regarding the distance through which the detonation acts. In regard to translation of the explosive force into mechanical terms, we have calculations which show that the total charge in a No. 6 Electric Blasting Cap produces detonation forces equivalent to 477 gram calories per cap."

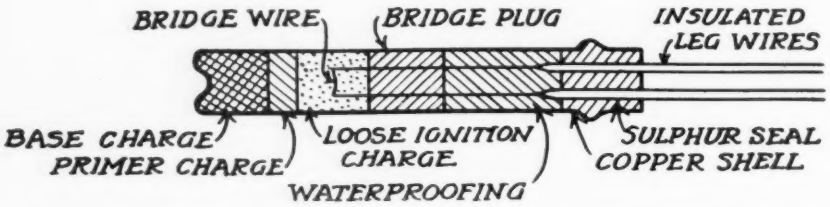

Fig. 2.—Longitudinal section through the du Pont No. 6 electric blasting cap.

METHODS

Adult dogs weighing between 6 and 20 kg. were used in all experiments. Preliminary examinations included gait, ability to jump, eye grounds, reflexes, placing and hopping reactions, cardiac and respiratory rates, rectal temperature, and femoral blood pressure. The cerebrospinal fluid pressure was measured by inserting a needle in the cistern after novocainization following the method described by Webster and Freeman.[4] When measurements were repeated, twenty-four hours were allowed to elapse between repetitions. The femoral blood pressure was measured with the usual laboratory mercury manometer after inserting an 18-gauge needle into the artery.

Prior to inflicting the trauma, the animals were lightly anesthetized with ether for from three to six minutes. The anesthetic was then discontinued, several turns of bandage wound about the head, and the blasting cap fixed to the desired location with adhesive tape. The dog was laid on its side and the head placed in an ordinary bucket to prevent the scattering of bits of copper. The cap was then fired. Respiration ceased immediately with the chest in inspiratory apnea, and the usual reflexes could not be elicited. In ten to ninety seconds respiration was resumed, either spontaneously or after a few compressions of the chest. When prolonged artificial respiration was necessary, the animal almost

invariably died. Five animals were not anesthetized. In all animals, a scalp laceration about 4 cm. in diameter occurred. Bleeding was stopped immediately by a pressure bandage and suture of the scalp. The wound healed in two to three weeks. In surviving animals, there was no evidence of skull fracture; an occasional animal killed outright by the force of the explosion showed a linear fracture across the longitudinal axis of the vault. For the first three to four days following the injury the surviving animals refused food and water and were tube fed daily with 500 to 1,000 c.c. of milk.

Two precautions were necessary in placing the blasting cap: (1) The cap should be taped securely to the head, or no effect could be expected on the animals. (2) The cap should not be placed over the frontal region of the head, because it caused depression fractures into the frontal sinuses accompanied by paraplegia, which did not occur in closed head injury. Aside from these precautions, it made little difference what areas were chosen for placing the cap, since the same neurologic disorder was always obtained. The sites best suited for placing the cap were the external occipital protuberance, the area just above the zygoma, and the parietal areas.

RESULTS

1. *Fatalities.*—

Immediate Death: Of thirteen animals which died as a direct result of the trauma to the head, nine died immediately. In these dogs respirations ceased and never returned, and after a short variable interval of five to ten minutes, the cardiac pulsations could no longer be heard, even though artificial respiration was maintained. During the interval between cessation of respiration and heart beat, all reflexes—corneal, pupillary, patellar, withdrawal—were absent. Gross examination of the brains in this group showed a varying degree of trauma as judged by the amount and location of hemorrhage. Three animals showed an insignificant degree of subarachnoid bleeding without any evidence of hemorrhage in the nervous substance; in two there was marked intracerebral hemorrhage; the remainder commonly showed petechial hemorrhages (one to five in number) usually in the brain stem and occasionally in the cerebral hemispheres (Fig. 3).

Postponed Death: Four animals showed what might be termed a postponed death. After an initial stage of apnea and areflexia, spontaneous respiration was resumed and the reflexes returned. The animals were flaccid and appeared to be in deep coma. The respirations were slow and shallow. Aside from occasional blinking no active movements occurred. In one-half to one and one-half hours respirations ceased. Shortly prior to death the pupils became fixed and the corneal reflex disappeared, although the patellar and withdrawal reflexes could still be elicited. Delayed deaths in the clinical sense, that is occurring twenty-four to forty-eight hours after head injury, were never observed

Fig. 3.—Immediate death. Cross section through the brain at the level of the optic thalamus and through the medulla oblongata at the level of the cochlear nuclei. Hemorrhages[11] into the reticula substance of the latter and in the doro medial part of the left optic thalamus.

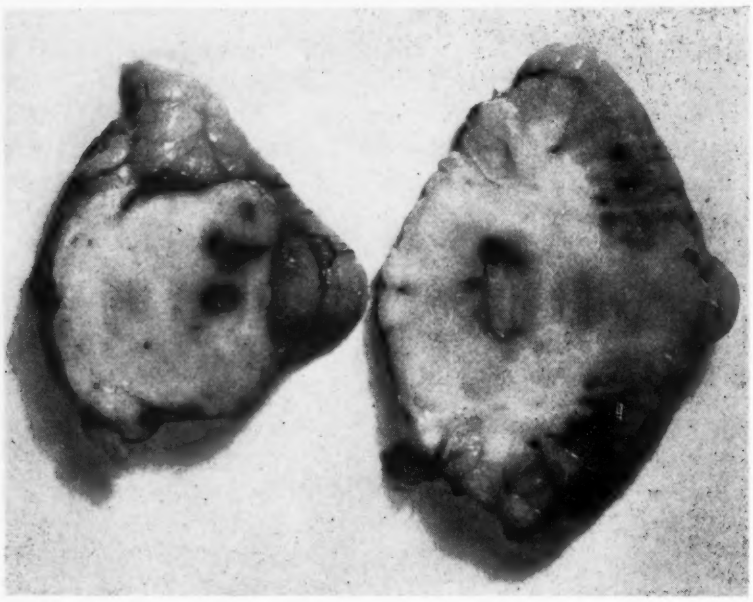

Fig. 4.—Postponed death. Numerous pin-point hemorrhages into the cerebellar hemispheres and into the medulla oblongata. Larger hemorrhages into the anterior and posterior colliculi extending into the aqueduct.

in this study. At necropsy, two animals showed a minor degree of subarachnoid blood without involvement of the brain substance. In the others, a variable amount of hemorrhage into the nervous substance was found with petechial hemorrhages into the brain stem (Fig. 4).

Fig. 5.—Surviving dog. Hemorrhagic contusion by direct impact of the blast on the right occipital lobe close to the surface. Hemorrhages into the temporal lobe and in the internal capsule at the level of the optic thalamus.

2. *Surviving Animals.*—Seventeen dogs survived the immediate effect of the trauma. Five of these died of unavoidable complications, namely one on the fourth day following the accidental aspiration of milk; one on the third day, due to meningitis; and three on the seventh, eighth, and tenth days, respectively. The latter were found dead in their cages after apparent improvement, and no cause for the death could be found at necropsy. These deaths were probably not related to the head trauma, since they occurred during a heat spell at a time when deaths among

normal animals were not infrequent. Most of the remaining twelve animals were sacrificed between the fifteenth and twenty-fifth day after trauma. Two animals were still being observed eight months after injury.

The surviving dogs presented a syndrome characterized by somnolence, inability to stand, ataxia, and salivation in the early phase, and by difficulty in jumping, disturbance of gait, and tendency to circle walking in the final phase. The early phase lasted from one to seven days, the final phase, for the duration of the observations.

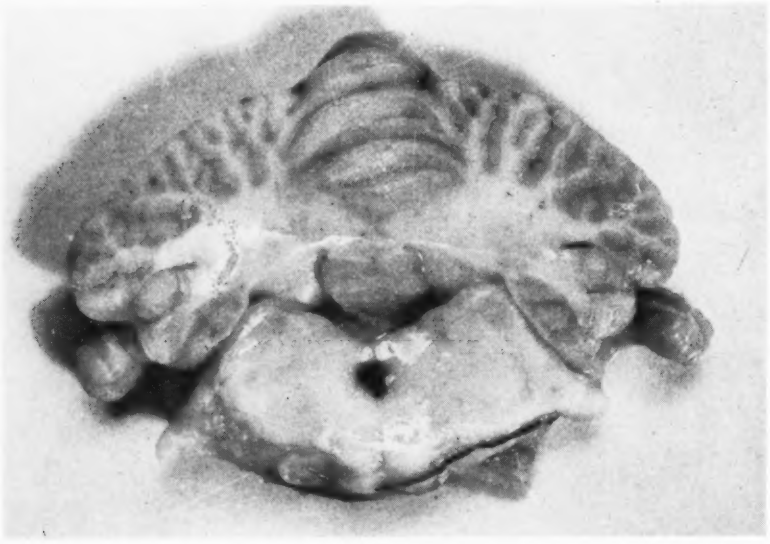

Fig. 6.—Surviving dog. Hemorrhage into the midline of the medulla oblongata at the level of the cochlear nuclei.

The sequence of events following detonation was: The corneal reflex disappeared for a period of from ten to ninety seconds and the animals became apneic. Respiration was resumed spontaneously or after a few compressions of the chest. After five to ten minutes, blinking returned. For the next several hours, the majority of dogs lay practically motionless on their side. If put on their limbs, they slumped. Occasionally they lifted the head for short intervals, or made feeble movements with the limbs. When the head was lifted passively and released, it fell with a thud to the floor. For the most part the eyelids were kept closed as if in sleep, but were opened when the fur was ruffled. Loud noise, as bursting a paper bag or dropping a bucket beside the head, produced no response. Drooling frequently persisted for forty-eight hours. Obnoxious odors, as ether or cigarette smoke, caused withdrawal of the head.

A second group of dogs showed, in contrast to this picture of inactivity, pronounced incoordinate motor hyperactivity. In an effort to stand, they thrashed about wildly, leaped at times fully one foot from

the floor and fell heavily. When exhausted, they wallowed confusedly, or tossed the head aimlessly and bewildered from side to side. Saliva drooled from the mouth, and often a coarse nodding head tremor was present. Sometimes they crawled on flexed limbs. Between periods of violent activity, they dozed quietly, but if aroused by being placed on their limbs, their wild ataxic struggle was resumed. These movements had no definite pattern, and rarely showed the irritative signs of rolling, circling, or gyration which are described following labyrinthectomy. Except for the first hour following the trauma, nystagmus was conspicuously absent. After five to six hours, this motor activity disappeared gradually and the picture resembled that of the first group of animals.

In one to seven days, usually two to four, the animals regained the ability to stand. The gait was halting, the dog tottered and fell frequently. By the end of a week all animals presented an almost uniform picture which persisted for many months, probably permanently. They walked with a broad base, more noticeable in the hindlimbs. The head was usually kept downward, pointed to the floor, tilted and rotated somewhat toward the blasted side. Often they stumbled or reeled. They would not stand on their hindlimbs to obtain food; in some instances, when such an attempt was made, the dog fell backward. When fed by hand, a slight but distinct head tremor was present. The dogs were not agile in retrieving thrown objects; often the object was not found unless it was rolled slowly and not too far. Some animals walked at times in circles, predominently toward the blasted side. This movement occurred in all animals after blindfolding. In "shuddering," the movement began in the head and forebody, increased in intensity as it progressed backward, and ended with a distinct flip to the rear. All animals were unable and refused to jump from distances as low as one to two feet, falling awkwardly when the attempt was made.

Neurologic Examination.—Except for transient minor deviations from the normal shortly after the blast, the examination showed normal hopping and placing reactions, adequate pupillary, corneal, patellar, withdrawal, and tonic neck and body reflexes. The eye grounds were normal. Nystagmus, although frequently observed early after injury, seldom persisted and was not a prominent feature. None of the animals was blind. No definite conclusion could be reached as to the degree of hearing impairment, although it was felt that the animals were probably deaf in the early phase. Hemorrhage from the ear was not observed and the drum heads were always found intact at necropsy. A few dogs showed bloodstained mucus in the nostrils; infrequently, ecchymoses of the ocular conjunctiva were observed. The cardiac rate was frequently slowed to a range between 60 to 70 beats per minute, which persisted for two to three days and gradually increased with the resumption of activity. When slowing of the pulse occurred, it ap-

peared within several minutes after the injury. The femoral blood pressure and the rectal temperature remained remarkably constant and showed no deviation from the pre-experimental studies. The cerebrospinal fluid was blood tinged and contained from 2,000 to 5,000 red blood cells on the day of injury, clearing in about four days. A moderate elevation of pressure was consistently observed. This ranged from 180 to 325 mm. of water pressure as compared to pretraumatic readings of 90 to 160.

Further neurologic examination showed that the difficulty in jumping so persistently present in surviving animals was caused by the absence of the labyrinthine righting reflexes. When a normal blindfolded dog held by the pelvis suspended in mid-air was suddenly released, the limbs become extended and the animal landed on its feet. In the traumatized dogs, this reflex extension of the limbs did not take place and the animal landed on its belly, even when not blindfolded. In three animals which were tested, another consistent neurologic sign was the absence of after-nystagmus following rotation in a Bárány chair. These animals accepted food immediately after rotation, suggesting an absence of vertigo.

The gross necropsy findings of the brain in this group of animals again showed a lack of uniformity. In four brains there was no evidence of contusion or hemorrhage. Eight animals showed a superficial contusion of the cortex at the site of impact, although the skull and dura remained intact. In two, this was the sole demonstrable lesion. The remaining six showed, in addition to the local trauma, one to five small hemorrhages, usually in the brain stem, infrequently in the hemispheres. In one animal, no contusion of the cortex was found, the only gross lesion seen was a petechial hemorrhage in the midline of the midbrain in the region of the brachium conjunctivum.

DISCUSSION

These results indicate that the labyrinthine system is consistently involved in all dogs surviving our experiments. This syndrome, characterized by an absence of postrotatory nystagmus, impaired labyrinthine-righting reflexes, circle walking, and inability to jump, has not been described, as far as we know, in the literature on experimental head trauma. Duret (1878)[9] occasionally noted motor incoordination and ataxia following experimental head injury in dogs, which he termed "locomotor ataxia traumatique." He felt that these signs were connected with hemorrhages in the upper cervical cord. Jakob (1912)[7] seems to have observed at times a similar picture in rabbits as the result of repeated blows upon the head. He concluded, however, that there was no relation between the severity and frequency of the trauma, on the one hand, and the consequent functional and pathologic disturbances, on the other.

Although a constant physiologic disturbance followed the standardized trauma in our experiments, the amount of damage to the brain was variable. The most frequent gross lesions found were small hemorrhages into the brain stem, occurring in half of the animals. A few animals, even upon microscopic examination of the brain (to be reported in a later article), showed only insignificant alterations from the normal. The syndrome, therefore, cannot be attributed to a damaged brain. Rademaker[10] and others have shown that the centers for the labyrinthine-righting reflexes are in intimate association with those of the body-righting reflexes, and that these centers are located in or about the red nucleus. In our animals the body-righting reflexes were normal. It is difficult to conceive of gross trauma applied to almost any portion of the skull constantly producing selective destruction of certain nerve cells leaving adjacent centers intact. The loss of postrotatory nystagmus again points to the labyrinth as the organ damaged. Persistent loss of the ability to jump with loss of postrotatory nystagmus is known to follow labyrinthine destruction in dogs.[11] Dow[12] has shown that the vestibular-righting reflexes are similarly and permanently lost following labyrinthectomy in primates, the effects being most marked in the monkey and least in the chimpanzee. Here, again, the most prominent finding, in addition to the loss of postrotatory nystagmus, was the absence of extension of the limbs when the animals were allowed to slide down an inclined plane. "Animals without labyrinths, lacking such extension when blindfolded, fell away from the inclined plane, and at times rolled completely over as an inert mass." Although the labyrinthine lesions have not been verified histologically, in our animals, the evidence strongly points to these organs as the site of damage.

Reports in the literature presenting evidence of damage to the labyrinth in man following head injury are abundant. This literature has been ably reviewed by Linthicum and Rand[13] and need not be restated here. Brünner,[14] using repeated blows upon the heads of guinea pigs as the method of trauma, found hemorrhage, exudates, and edema in various portions of the inner ear, even though no gross abnormality in muscular coordination of the animals was reported. He confirmed these pathologic findings in human necropsies following deaths from head injuries. Linthicum and Rand, discussing their observations in patients following concussion of the brain, found that "in practically all cases of cerebral concussion the patient complains of some form of equilibratory disturbance," and this disturbance could be demonstrated by adequate neuro-otologic examination. More recently Russell[15] expressed the opinion that ". . . true vertigo which often follows head injuries is probably due to concussion of, or hemorrhage into, the labyrinth." He emphasized the importance of this type of damage as a cause of incapacity following head injury.

The role of the labyrinths in the clinical condition designated as "cerebral concussion" is unknown. One might speculate as to whether

the clinical symptoms, both early and late, could be attributed to sudden cessation of function of these organs with partial or complete recovery. It is conceivable that concussion may result from widespread inhibition of the central nervous system acting through a reflex, possibly originating in the labyrinths. This theory is supported by investigations dealing with the role of head acceleration in experimental head trauma. Denny-Brown and Russell[3] found that "concussion" ensued when the degree of trauma applied to the freely movable head reached a critical acceleration of 28 feet per second. When the head was fixed, far greater forces were necessary to produce "concussion." We have estimated the head acceleration of our dogs, by means of rapid cinematographic films taken at the time of the explosion, to be in the neighborhood of 64 feet per second. Under these experimental conditions, fixation of the head did not alter the results. The trauma produced by the blasting caps was obviously of much greater force than that reported by Denny-Brown and Russell. Hence, their findings are not contradicted by our experiments. Although these observers were inclined to disregard the role of the labyrinth in concussion, they presented no evidence in favor of their conclusion. If, in mild head traumas, acceleration is an important factor, and in severe head traumas labyrinthine function is lost, the inference is that the labyrinth may play a greater role in closed head injury than heretofore suspected. Crucial experiments must be devised to determine the actual role of the labyrinth in concussion.

SUMMARY

1. A method of producing a severe type of standardized closed head injury in the dog with blasting caps is described.

2. Surviving dogs presented a uniform neurologic syndrome. In the first days, this was characterized by somnolence, inability to stand, ataxia, and salivation. Permanent disabiltiy consisted of inability to jump, tendency toward circle walking, absence of postrotatory nystagmus, and impaired labyrinthine-righting reflexes.

3. The gross necropsy findings of the brains in all animals showed a lack of uniformity, indicating that the hemorrhages present were not responsible for either death or the neurologic syndrome. In some animals no hemorrhages were found. Small hemorrhages into the brain stem were found in half of the brains.

4. The evidence suggests that the disturbance uniformly produced by the blasting cap was due to damage to the labyrinths.

REPRESENTATIVE PROTOCOLS

Immediate Deaths.—

PROTOCOL I.—DOG 426. Male. 12.2 kg.

5/1/42. 8:42 A.M. Ether administered for four minutes. One cap taped over right parietal region.

8:48. Cap fired. Respiration stopped. Chest in inspiratory apnea. Pupils dilated and fixed. Tendon reflexes absent. Artificial respiration started.

8:50. Pulse 126. Still apneic.

8:54. Pulse 168.

8:58. Heart sounds no longer audible.

5/1/42. Necropsy: Viscera normal. Tympanic membranes intact. Right temporal muscle and scalp lacerated. Skull intact. There was a thin layer of blood over the entire right hemisphere and the posterior part of the left hemisphere. No hemorrhages in the brain.

PROTOCOL II.—Dog 960. Male. 13.4 kg.

6/15/42. One cap taped to external occipital protuberance.

2:34 P.M. Cap fired. Thrown violently to the floor. Respiration ceased, all reflexes absent. Artificial respiration started.

2:40 Pulse over 200.

2:42. Dead.

6/15/42. Necropsy: Visceral normal. Tympanic membranes intact. Scalp lacerated. Temporal muscle normal. Skull intact. Slight amount of subarachnoid blood. The brain appeared normal except for moderate congestion of the veins. Cross section showed nothing abnormal except for moderate enlargement of both lateral ventricles from the middle of the ventricle backward extending downward into the inferior horn. The left ventricle was larger than the right. In the pons there were several minor and one major hemorrhage into the basal part.

Postponed Deaths.—

PROTOCOL III.—Dog 113. Male. 6.8 kg.

6/17/42. One cap taped over right zygoma.

2:33 P.M. Cap fired. Thrown to the floor. Apneic. Pupils dilated and fixed. Corneal reflex absent. Artificial respiration started.

2:42. Breathing spontaneously, very shallow, 12 per minute. Pulse 12. Corneal, patellar, and withdrawal reflexes still absent.

2:46. Pulse 12. Respirations 9. Pupils fixed. Corneal reflex sluggish. Patellar and withdrawal reflexes present. Limbs flaccid.

2:51. Occasional blinking. Pulse 7. Respirations 8, very shallow.

2:56. Pads of limbs cold. Respirations 4.

2:58. Artificial respiration resumed. Pulse 10.

3:03. Pulse 10.

3:06. Pulse 36.

3:09. Animal dead.

6/17/42. Necropsy: The right temporal scalp was lacerated. The temporal fascia was intact and the muscle was not injured. There were no fractures. There was a slight amount of blood in the subarachnoid space over the hemispheres. The surface of the brain was entirely normal except for possible bruising of the right flocculus. Cross section failed to reveal any pathology except for the very slight superficial lesion of the right flocculus.

PROTOCOL IV.—Dog 468. Male. 16.6 kg.

6/16/42. 11:08 A.M. Ether for five minutes. One cap taped to external occipital protuberance.

11:15. Cap fired. Respiration stopped for 40 seconds. Corneal reflex absent for one and one-half minutes. Pulse 39.

11:29. Respiration 38, rapid and shallow. Pulse 39. Forelimbs slightly rigid. Tendency to opisthotonos. Unable to stand, or even to raise head. Pupils moderately wide, reacted normally to light. Patellar and withdrawal reflexes present.

1:00 P.M. Lays motionless on side. All limbs flaccid. Salivation profuse. Corneal reflexes absent. Pupils fixed. Pulse 82. Respiration 10, very shallow.

1:08. Pulse 104. Respiration 8.

1:11. Artificial respiration started.

1:15 Dead.

6/16/42. Necropsy: The left temporal muscle was lacerated. There was a stellate fissuring of the vault radiating from the external occipital protuberance, but no depression of the fragments. The dura was intact. There was a slight collection of blood beneath the dura, more marked on the left. The base of the skull was intact. The brain showed mild subarachnoid hemorrhages over the two pyriform lobes, over the medial lobes of the convexity, and contusion of the left parieto-occipital lobe. Cross section showed a hemorrhagic infarct of the parietal lobe on the left side, the site of injury, extending backward almost to the tip of the occipital lobe and involving the underlying cerebellar hemisphere and flocculus. There is a pinhead-sized hemorrhage at the typical spot in the brachium conjunctivum extending downward and backward to the trapezoid body.

Surviving Dogs.—

PROTOCOL V.—Dog 430. Male. 14.0 kg.

5/10/42. Ether for five minutes. One cap taped just to the right of the external occipital protuberance.

11:32 A.M. Cap fired. Respiration stopped for 30 seconds. Resumed spontaneously.

11:35. Whining, moving head slightly.

11:50. Made no effort to move head or limbs. Limbs flaccid. When head was raised passively and released, it dropped with a thud. Corneal reflexes sluggish, but present. Pupils equal, moderately dilated, reacted briskly to light. Fundi normal.

12:30. Could barely raise head from floor. Slow, nodding head tremor. Placed on feet, slumped immediately and made a few feeble movements with the limbs.

2:00 P.M. More active. Several unsuccessful attempts to stand. Body-righting reflexes normal. Flexing and extending head gave normal response upon limbs. Sliding body along the floor caused elevation of the head. Twisting of the body was followed by turning of the head in the corresponding direction. Twisting of the head was not followed by turning of the body.

5/10/42. 10:00 A.M. In general drowsy with little activity. Head tremor still present. Twice within last hour was seen to crawl a few steps on flexed limbs. Placed on feet, limbs slid outward and the animals sprawled awkwardly.

5/12/42. Unable to stand. Held head up for longer periods of time. Retracted head from ether or cigarette smoke. Head tremor not as noticeable as previously.

5/13/42. Unchanged.

5/14/42. When placed on feet able to stand on a broad base for a few seconds. Fell on the attempt to walk.

5/16/42. Walked on broad base, fell frequently. Ate spontaneously.

5/18/42. Walking much better. Slight head tremor when reaching for food.

5/21/42. Walked on broad base. Frequently circled to the right. Shuddered frequently, ending with a flip of the buttocks. Unable to jump from a distance of one and one-half feet without falling. Placing and hopping reflexes and all postural reactions were normal with the exception of the labyrinthine reflexes.

5/26/42. No change.

5/27/42. Sacrificed. Viscera normal. There was a small laceration of the right scalp almost completely healed with some contusion of the underlying temporal muscle. Both the base and the vault of the skull were intact. The dura appeared normal except over the right occipital lobe where there was a bluish discoloration. In this region the dura was firmly adherent to the underlying cortex. The brain showed hemorrhage, probably contusion, over area 19 of the right occipital lobe,

about 1 cm. in diameter, but extending to a minor degree to the medial, as well as the posterior margin of the hemisphere. There was some minor hemorrhage at the basal surface of the brain at the angle of the entering right eighth nerve. The pia at the base of the brain overlying the chiasmatic cistern was opaque and slightly thickened. On cross section, small hemorrhages, the size of a pin point, were found within a distance of 1 mm. of each other in the medial aspect of the left third temporal convolution and the bordering lateral aspect of the basal longitudinal gyrus at the level of the ascending fornix. There were two other bleedings of the same small size in the left hemisphere, one below the left anterior commissure and the other in the middle of the corpus striatum. There were larger hemorrhages in the middle of the left temporal lobe bordering the hippocampus and a fresher focus, possibly a thrombosis, at the same level in the left internal capsule. There was a bilateral, slight but definite enlargement of the lateral ventricles. Sections through the occipital hemorrhage showed the involvement of the cortex as expected. A brown discoloration was seen in the entering root of the right eighth nerve. No other gross lesion was found in the brain stem.

PROTOCOL VI.—Dog 478. Female. 9.2 kg.

5/7/42. Ether for four minutes. One cap taped to the external occipital protuberance.

1:24 P.M. Cap fired. Apnea for 60 seconds, respiration resumed a few compressions of chest.

1:26. Whining, raised head. Pupils dilated, reacted sluggishly.

1:37. Attempted to stand, fell. Lying on belly with limbs sprawled and head upright. Tossed head from side to side. Head tremor.

1:58. Crawled a few steps on flexed limbs.

3:37. Asleep. No response to loud noise. When grasped, eyes opened, animal righted itself spontaneously and crawled a few steps.

5/11. Able to stand for a brief interval. Fell on the attempt to walk.

5/13. Unchanged.

5/14. Walked hesitantly on broad base staggering noticeably. Responded to call. Walked in circles mostly to the left, at times to the right.

5/18. More active. Gait improved. Still walked drunkenly, slipping and falling at times.

5/20. Gait unchanged. Walked in circles, mostly to the left, at times to the right. In jumping from a height of one and one-half feet, fell clumsily. Placing and hopping reactions normal. Tonic neck and body reflexes normal. Labyrinthine righting reflex absent. No postrotatory nystagmus.

5/22. Necropsy: Viscera normal. Scalp healed. Skull intact. Meninges normal. Symmetric subpial hemorrhages about 1 cm. in diameter covered the outer surface of the two occipital lobes. On frontal sections a small area was suspected of being a softening at the level of the descending fornix in the lateral angle of the internal capsule and the temporal lobe. The lateral ventricles were slightly enlarged. The right lateral ventricle was, in its posterior part, a trifle larger than the left. In the medulla oblongata, a hemorrhage the size of a pinhead was located at the level of the entering vestibular nerves in the midline, 2 mm. below the ventricular surface and in or immediately below the posterior longitudinal fasciculus.

PROTOCOL VII.—Dog 428. Male. 18.8 kg.

5/6/42. Ether for six minutes. One cap taped to the external occipital protuberance.

8:02 A.M. Cap fired. Respirations stopped 40 seconds. Resumed spontaneously.

8:09. Lying motionless. Limbs flaccid. Pupils wide, reacted sluggishly. Withdrawal, patellar, and corneal reflexes present.

8:15. Blinking. Profuse salivation.

8:20. Raised head. Rolled onto crouched limbs. Attempted to stand, fell heavily. Repeated efforts several times in rapid succession with increasing violence. At times leaped fully a foot from the floor. Panting heavily. Glaring, tossing head from side to side. Seemed confused. Head tremor.

9:30. Lying on left flank, head between forepaws, eyes closed. Drooling profusely. No response to noise. When touched, eyes opened. Placed on feet, resumed violent movements in an attempt to stand.

12:06. Quiet for the most part. Occasional attempts to stand. Could crawl on flexed limbs.

3:00 P.M. Ataxic attempts to stand could be elicited by placing dog on feet. The intensity of the movements were diminishing.

5:00. When placed on limbs, fell limply. Ataxic movements no longer could be elicited.

5/7. Little activity. Nodding head tremor still present to minor degree. When placed in upright position stood on a broad base, did not attempt to walk, but lay down after a few moments. With hindlimbs held, could walk on forelimbs; able to stand on hindlimbs with tendency to give way in one or the other limb. In supine position, with head extended, all limbs extended headward; with head flexed, limbs relaxed. Rotation of body followed by head rotation; turning head followed by twisting of body.

5/8. Definitely better. Walked drunkenly and soon fell.

5/10. Walked the length of the room, slowly, hesitantly on a broad base.

5/13. Unchanged.

5/15. More active. Walked on broad base, swaying drunkenly at times. Responded to call. Shudder ended in tail flip; at times this movement threw the animal to the floor. Walked in circles to the left. Refused to jump from height of two feet, prefering to remain on table. When pushed, fell clumsily.

5/20. Extremely restless. When out of cage, walked about continuously, stopping only for a moment or two to scratch or to shudder. Circle movements to the left were frequent. Unable to jump without falling. Labyrinthine-righting reflexes and postrotatory nystagmus absent.

5/25. Sacrificed. Viscera normal. Ear drums normal. Skull intact. Dura normal. The surface of the brain showed nothing abnormal, nor did frontal sections, with one questionable exception at the level of the red nucleus. The surface here seemed to be sprinkled with enlarged blood vessels or small hemorrhages in the brain stem, while the region of the claustrum was somewhat reddish.

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A CASE OF INFECTION WITH CLOSTRIDIUM SORDELLII AND A CASE OF GAS GANGRENE TREATED BY PENICILLIN

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TWO cases of mixed infections with anaerobic microorganisms are reported because of certain features of interest attached to them. The first patient was infected by *Clostridium sordellii* and *Clostridium welchii* and was treated with penicillin. The second developed gas gangrene of the abdominal wall following an operation on the biliary tract. A third case mentioned is so similar to the second that the two may supply a syndrome of this unusual complication. The second patient was treated with penicillin and polyvalent antiserum, the third received no specific treatment. Although all three patients died, no general conclusion can be drawn from them concerning the efficacy of penicillin in similar infections. Many cases will have to be collected, treated at various periods in the course of their infection, and with various methods of administration, before evaluation of penicillin therapy can be made. Because these cases are relatively rare and because no one sees many of them, they are put on record.

CASE 1.—A 30-year-old American born Negro woman had been treated in the outpatient clinic for asthma and recurring head colds which were thought to be allergic in origin. Two days before admission to the hospital, because of an asthmatic attack she received from a doctor on the outside a hypodermic injection of adrenalin, morphine, and atropine in the back of the left arm. The following day she was aware of swelling and tenderness in the region of the injection. These symptoms increased, and she became feverish. Shortly before admission to the hospital she noted rapid increase of the rate of swelling and she experienced periods of great prostration. The significant physical findings, treatment and course were as follows.

May 31, 1943, the patient was admitted at 8:30 P.M., appearing ill. Rectal temperature was 102.6° F.; pulse, 76; respirations, 20. There was great swelling of the left upper extremity from the upper deltoid region almost to the elbow. The swelling extended around the upper arm so that it had to be held away from the body, and also involved the axilla. It was so tense that it suggested to the first doctor who saw her the possible need for releasing incisions through the deep fascia. There was some tenderness in the swollen area, most marked on the medial and axillary aspects. The lack of redness and heat was striking. There was no fluctuation. The radial pulse was difficult to feel at either wrist because her blood pressure remained low, at times being almost unobtainable. Urine showed albumin 3 plus. The blood count was white blood cells, 30,750; polymorphonuclears, 86 per cent (0–17–69); lymphocytes, 11 per cent; monocytes, 1 per cent; eosinophiles, 2 per cent.

Treatment that night consisted of high elevation of the arm, poultices to the upper arm, sulfadiazine 1 Gm. every four hours with soda bicarbonate 0.5 Gm.,

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amytal 0.1 Gm. repeated once, codeine, 0.06 Gm. and aspirin 0.6 Gm. repeated twice. She slept fitfully, complaining of pain in the swollen area, and generalized itching. At times she seemed to be irrational.

June 1, 8 A.M., the temperature was 99.8° F.; pulse, 96; respirations, 22. The swelling of the arm had extended throughout the forearm to the wrist. The blood pressure was hardly obtainable, although the patient was clear mentally. The medical consultant thought that the swelling could not be explained upon an allergic basis. X-ray of the arm showed no gas in the tissues. There were still no signs of a focus of infection. The sulfadiazine was stopped after she had received 3 Gm. in twelve hours.

Operation.—Incision 10 cm. in length under local novocain anesthesia in the posterior aspect of the arm, where the hypodermic injection had been given which was the probable source of the infection. The subcutaneous tissues were extremely firm and grayish yellow in color. Clear watery fluid exuded when the tissues were divided. There was practically no bleeding. The patient went into shock just before the operation. The tissues were divided through the brachialis fascia without encountering pus or gas. The underlying muscle appeared normal. The wound was tamponed open with China silk.

Pathologic examination of a specimen of the subcutaneous tissue was later reported to show scattered small foci of lymphoid infiltration, but nothing suggestive of acute inflammation. Culture of a specimen of the subcutaneous fat showed no growth, but *Cl. welchii* and *Cl. sordellii* appeared next day from swabs moistened in the wound at the same time. There was no aerobic growth.

Eight hundred cubic centimeters of pooled plasma and 700 c.c. of saline solution were given by infusion about the time of the operation.

The swelling of the arm increased in spite of profuse discharge of serous fluid from the incision. An hematocrit taken shortly before midnight was 57.8, plasma specific gravity 1.0217, plasma protein 5.03. The blood in the tube was hemolyzed.

June 2, 12:30 A.M., further multiple incisions through the deep fascia were made into the area of swelling, because of the extreme tension of the tissues. Her blood pressure was at this time unobtainable. Pulse was about 100. Five hundred cubic centimeters of whole blood were given. A generalized urticaria followed. At 3 A.M. 300 c.c. of plasma were given, without reaction. Pulse during the night remained at about 80 to 88. Temperature went to 102.4° F. but at 8 A.M. was 100°. Hematocrit at this time was 73.7, plasma specific gravity 1.0223, plasma protein 5.24. The blood sample was hemolyzed. Blood count was red blood cells, 6,950,000; hemoglobin, 19.4 Gm. (134 per cent); white blood cells, 34,650; polymorphonuclears, 94 per cent (1–47–46), lymphocytes, 5 per cent; monocytes, 1 per cent. At 12:30 P.M. 500 c.c. of plasma and 500 c.c. of saline solution were given by infusion. At 1:30 P.M. soon after microorganisms resembling *Cl. welchii* were reported in the culture from the wound, 50,000 units of sodium penicillin were given intramuscularly. At 2:45 P.M. 50,000 more units of penicillin were given intravenously, and this was repeated at 4 P.M., and again at 9:45 P.M. At 3:45 P.M. hematocrit was 75 and plasma protein 3.92. At 9:15 P.M. she received 500 c.c. of plasma. The total fluid intake during this day was 6,050 c.c. and urine amounted to 350 c.c. There was much serous discharge from the wounds. Late in the day the patient's temperature rose to 102.6° F. at which time her pulse was 80 and her blood pressure unobtainable. At 11 P.M. hematocrit had fallen to 67.5 and plasma protein had risen to 4.52.

June 3, 1943, at 1 A.M., she received 350 c.c. of plasma containing 50,000 units of penicillin, and two hours later 300 c.c. of plasma. Her pulse about this time dropped below 60. She seemed quite alert but not wholly oriented. Ten thousand units of penicillin were given intramuscularly at 4 A.M., 7 A.M., and 10 A.M. Blood count, taken about 9 A.M., was red blood cells 7,550,000; hemoglobin 22.2 Gm.

(153 per cent); white blood cells 58,200; polyphonuclears 84 per cent (1–44–39), lymphocytes 16 per cent. Her temperature rose during the morning to 104.8° F. It was about this high at the time of death at 11:15 A.M. She had recognized her sister one-half hour before.

Post-mortem examination revealed the following significant abnormal findings: swelling and edema of the subcutaneous tissues of the left upper extremity with extension into the left side of the thorax and neck. In one region of the left pectoralis muscle there were many areas of necrosis and hemorrhage. There were 200 c.c. of clear yellow fluid in the peritoneal cavity, 250 c.c. in the right pleural cavity, and 750 c.c. in the left, with partial atelectasis of both lower lobes. Microscopic sections of the subcutaneous fat of the left arm showed edema, many small hemorrhages, perivascular accumulations of polymorphonuclear leucocytes which were undergoing degeneration, and a pale homogeneous coagulum within the walls and the lumens of some of the vessels. Gram-positive and gram-negative rods were observed in one area. *Cl. sordellii* and *Cl. welchii* were grown from the operative wounds and the latter also from the pectoral muscle, subcutaneous fat, and the right ureter.

The course of this patient's infection was rapid and little affected by treatment. Approximately twelve hours after receiving the fatal injection, she noticed swelling. Within forty-eight hours of the injection, she came to the hospital. The swelling at that time extended from upper deltoid to elbow, was very marked where it existed, and had well-defined borders. The nature of her condition was not at first recognized, and there was even some doubt whether she had an infection or an allergic reaction because of the absence of redness and heat in the swollen area, the intermittent and low grade of fever, and the slow pulse. The x-ray showed no gas in the swollen tissues.

Within sixty hours after admission to the hospital, or approximately four and one-half days after receiving the injection, she was dead. During her course in the hospital, the striking features were the enormous local swelling, the intermittent and finally constant state of hypotension, the relatively slow pulse and absence of high fever, and the progressive and extreme hemoconcentration. Treatment during the sixty hours in the hospital included 3 Gm. of sulfadiazine during the first twelve hours, not enough to be a test of the efficacy of the drug, 2,750 c.c. of plasma and 500 c.c. of whole blood during the last forty-three hours of life, and 280,000 units of penicillin intravenously and intramuscularly during the last seventeen hours of life. She received no specific antiserum because the *Cl. sordellii* were not recognized either by the clinical picture or by the laboratory during her life.

No clear effect on the progress of her infection was observed as a result of this therapy and only temporary and relatively minor effects on the course of her hemoconcentration. Although *Cl. welchii* was grown from various parts of the body, the case was clinically predominately one of infection with *Cl. sordellii*. Reference is made to the cases described by Meleney, Humphreys, and Carp,[1] in which the characteristic features were the tremendous woody edema which spread with more or less rapidity from the focus of infection, the absence of

pus or gas when the tissues were incised, the presence of hard white subcutaneous fat from which there was a copious discharge of serous fluid, the prostration, and the fatal outcome. Studies of blood concentration were not included in that report. Infections of laboratory animals show that the microorganism characteristically does not grow in great numbers in the tissues nor spread very widely, but produces the edema and toxemia by diffusion of its toxin. There is little evidence of death of tissue even at the focus.

The only treatment at present known is large doses of polyvalent serum which includes principles of antisordellii toxin. Supportive measures are also necessary and possibly releasing incisions. The prognosis is bad, but might be better if early diagnosis were made. This would be possible only if the doctor in charge of the case had the condition in mind and recognized the clinical picture. By the time that *Cl. sordellii* is isolated from the lesion and identified, the infection may well have been fatal.

CASE 2.—A 50-year-old Finnish woman came to the hospital complaining of attacks of pain in the right upper quadrant, associated on occasions with chills, fever, and jaundice. Her past history was remarkable only for two partial thyroidectomies for nontoxic goiter. Physical examination revealed nothing unusual except the scar in the front of her neck, and a diastolic cardiac murmur. She stated that she habitually climbed four flights of stairs without dyspnea. There were no abnormal laboratory findings. A cholecystogram revealed no evidence of a gall bladder shadow. On July 7, 1943, she was operated on with a diagnosis of chronic cholecystitis, cholelithiasis with stone in the common duct. A right subcostal incision was made, dividing the right rectus muscle and part of the lateral oblique muscles. There were many adhesions in the right upper quadrant; no gall bladder was recognized, but there was a calculus measuring 3 by 1 cm. in the common duct. After the calculus was removed, a probe could be readily passed into the duodenum and into both hepatic ducts. The ducts were thoroughly irrigated with saline solution. The bile in the common duct was muddy, and was subsequently shown to contain *Cl. welchii*, *Bacillus coli*, and *Streptococcus viridans*. There was no evidence of an acute inflammatory process. Exploration of the abdomen revealed no other abnormality. The appendix was removed, a T tube was fastened into the common duct and a soft rubber drain placed alongside it, and the wound was closed about the drains in layers, by means of interrupted silk sutures.

For twenty hours the postoperative course was not thought to be unusual, except that the patient could not void and had to be catheterized. During the morning of the first postoperative day, however, it was noticed that the patient was lethargic, that there was a good deal of serous discharge from the wound, and that bloody drainage was coming from the rubber tube, although bile was coming from the T tube. There was also found to be a hard, painful, tender area above the wound extending up toward the breasts. All this was apparent less than twenty-four hours after operation. Pulse by noon had risen to 130 and temperature to 103.6 When the wound was dressed, the amount of discharge was noted to be excessive, but the odor was not thought to suggest anaerobic infection. There was an area extending up from the wound which was swollen, hard, tender, and quite sharply outlined, corresponding roughly to the extent of the upper portion of the right rectus muscle. The lower margin of the wound was not affected. The wound was opened, but it was not until some sutures were taken out of the anterior sheath

exposing the muscle that a little gas was seen bubbling up. A smear taken of the exposed muscle showed gram-positive rods which looked like *Cl. welchii*, and later were proved to be. (*Bacillus aerogenes* and a nonhemolytic streptococcus were also cultured from the wound.) An x-ray of the wound area taken on the way to the operating room did not reveal gas in the tissues.

The second operation was done about thirty-three hours after the first, and consisted of removal of the portion of the rectus muscle cephalad to the original wound. It was almost completely destroyed by gas gangrene, but no other area of invasion was recognized. The procedure took thirty minutes. The wound was packed wide open with zinc peroxide. Before this procedure, hematocrit was 44, plasma specific gravity 1.0264, plasma protein 6.63. Blood count was red blood cells, 4,620,000; hemoglobin, 12.5 Grams (81 per cent); white blood cells, 30,500; polymorphonuclears, 86 per cent (0–38–48); lymphocytes, 13 per cent; basophiles, 1 per cent. Before operation 50,000 units of penicillin were given intravenously, and 25,000 units at three-hour intervals until the patient died, a total of 175,000 units in sixteen hours. Starting about three hours after the second operation, she received polyvalent gas gangrene serum intravenously as follows: 60 c.c. at 1:15 A.M., 30 c.c. at 6 A.M., 60 c.c. at 10 A.M., 11 A.M., 1 P.M., 2:15 P.M., a total of 330 c.c. in thirteen hours. She also received 500 c.c. of plasma about the time of the second operation, 1,000 c.c. of saline solution by clysis shortly before the operation and 4,000 c.c. of 5 per cent dextrose in saline solution by vein during the night following operation. On the morning of July 9, ten hours following the second operation, venous pressure was 100 mm. and hematocrit, plasma specific gravity, and plasma protein essentially the same as they had been the day before. One liter of urine was collected by catheterization. Her temperature during the night fell dramatically from 106.6° F. shortly before midnight to 99.4° at 8 A.M. It did not again rise. Her pulse meanwhile fell from 130 before operation to 100 the next day, where it remained almost until she died. Nevertheless, her blood pressure fell, breathing became wheezy, and she began to cough after the second injection of anti-toxin and for that reason only half the dose was given. Subsequent doses of antitoxin did not have this effect, but breathing remained somewhat difficult and moist, and she became jaundiced. In spite of the drop in temperature and pulse, she looked more ill. She died at 3:40 P.M., fifty-two hours after the first operation.

At autopsy, virtually no evidence of active infection was found. No *Cl. welchii* were grown from cultures of the wound. The patient did have edema of the glottis and some edema of the lungs, and also a rheumatic heart with a rather tight mitral stenosis. The jaundice was unexplained. It was not due to persisting anaerobic infection, but may have been the result of the period of toxemia. The presence of bile plugs in the canaliculi suggested a factor of obstruction, but no cause of obstruction was found. The liver was not apparently the site of an infection. Incidentally, no gall bladder was found.

It would appear that the gas gangrene infection had been controlled, which might have been the result of any or all of the therapeutic measures taken, that is, the surgical removal of the focus, the zinc peroxide, the penicillin, and the serum. The patient's death might have been caused by any of a number of factors, such as the period of toxemia, too much intravenous fluid (although the hematocrit remained within normal limits), failure of a rheumatic heart, serum sensitivity, or edema of the glottis. It was probably a death due not directly to gas gangrene, but indirectly to it and to the chain of events which accompanied it.

CASE 3.—This case is briefly mentioned because of the similarity to Case 2. The patient was a 66-year-old woman who underwent cholecystectomy for acute cholecystitis. The incision was made transversely below the right costal margin, dividing the right rectus and part of the oblique muscles. The common duct was not explored because the woman had never been jaundiced. A rubber tube drain was placed along the bed of the gall bladder, and the wound closed around the drain in layers by means of interrupted silk sutures. Thirty-six hours after operation she complained of abdominal pain, and her temperature rose suddenly to 104° F. although her pulse at that time was only 106. Later it rose to 120. Respirations also went up to 28 and became grunting in character. Her condition was attributed to bronchopneumonia, which indeed she had, and the gas gangrene of the abdominal wall was not recognized until shortly before her death, which occurred sixty-two hours after operation and twenty-four hours after the sudden rise in temperature.

At autopsy, the gas gangrene was found to involve much of the abdominal wall below the incision. The time of onset shortly after operation, the rapid rise in temperature, the prostration and lethargy, the limitation of the infection to one edge of the wound, and the rapidly fatal outcome, were alike in Cases 2 and 3. The nature of the infection in Case 3 was not recognized in time to try penicillin or serum.

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SUBPARIETAL RUPTURE OF THE INTESTINE DUE TO MUSCULAR EFFORT

Case Report

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SUBPARIETAL rupture of the intestine due to external violence is not uncommon, but subparietal rupture of the intestine due to muscular violence is very rare. Up to 1937, Wilensky and Kaufman[1] were able to collect reports of 43 cases in which the latter type of injury occurred. Since that time cases have been reported by Aird,[2] Robinson,[3] MacMillan,[4] and Berman and Rosner.[5] Case 10 in the series of cases of abdominal injuries reported by Fairchild.[6] in 1931, should have been included in Wilensky and Kaufman's article. It is not unlikely that the cases reported by Mulkey[7] and Mufson[8] belong in this series.

Including the case herewith reported, a total of 50 cases have been recorded. All of the patients were men, and in at least 37 cases hernia was present. In only 4 cases was hernia definitely stated to be not present. Of the 50 patients, 28 died, a mortality of 56 per cent. Wilensky and Kaufman[1] found that the mortality in 22 cases in which operation was performed within twenty-four hours was 50 per cent and in 13 cases in which operation was performed after twenty-four hours had elapsed, 84 per cent. In 4 cases in which no operation was performed the mortality was 100 per cent.

The most plausible explanation of this injury is that the muscular effort increases the intraluminal pressure of the intestine, and if there is a weak point in the parietes, as at an inguinal hernia, a bursting rupture takes place (Bunge,[9] Haim,[10] and Bruce[11]), Aird[2] thinks that stretching the intestine between two fixed points causes its rupture. Tchistosserdoff[12] points out that adhesions make rupture more likely.

Patient A. B., an Italian workman aged 47 years, was admitted to the Evanston Hospital at 2:15 P.M., Oct. 20, 1943. At 9:30 A.M. of that day he was helping three other men lift a heavy gasoline engine when the weight shifted, giving him a heavy strain. He immediately experienced abdominal discomfort which was not severe, but by 11 A.M. the pain was so intense that he was unable to stand. He said that two or three months he had experienced "stomach-aches" after eating irregular meals.

On examination, tenderness and boardlike rigidity of the entire abdomen, perhaps more intense in the lower half, were noted. A right inguinal hernia was present. The leucocyte count was 10,000; temperature, 99.0° F.; pulse, 88; respirations, 22. The urinalysis was negative. X-ray examination failed to show pneumoperitoneum. A diagnosis of perforated peptic ulcer was made.

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The abdomen was opened through a right rectus incision at 3:35 P.M. of the same day. There was a considerable quantity of turbid fluid and intestinal contents free in the peritoneal cavity. The intestines, particularly in the lower half of the abdomen, were fiery red, and in many places there were patches of yellow exudate. A rapid search of the abdomen failed to show a perforation of the stomach, duodenum, appendix, or sigmoid colon. A perforation, 1 cm. in diameter, was found in the ileum about 60 cm. proximal to the ileocecal valve. The rim of intestine around the perforation was excised and sent to the laboratory of pathology for study. The opening in the intestine was closed transversely with a continuous chromic catgut Connell suture reinforced with fine silk mattress sutures. Sulfathiazole, 9 Gm., was placed in the peritoneal cavity, and the wound was closed without drainage. Postoperatively one-sixth molar sodium-r lactate was given intravenously to raise the pH of the urine. The excised rim of the perforation was reported to be normal intestine. The recovery was unusually smooth and uneventful save for a small abscess in the right rectus incision. On October 30, the inguinal hernia was repaired.

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AN ACCESSORY OPERATING TABLE FOR ORAL SURGERY ON INFANTS AND CHILDREN

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AFTER a limited personal experience in oral surgery and observations elsewhere, it seemed that the disposition of the patient undergoing cleft lip and palate repair, the arrangement of the surgeon, assistants, and anesthetist at the operating table, left much to be desired. The practice of placing the head of the patient on the knees of the surgeon or allowing it to hang over the edge of the table is not conducive to the maintenance of an adequate airway, to the stability of the operative field, or to the proper drainage and aspiration of blood, mucus, or excess ether vapor. With these points in mind a device was designed and built, and has been in use for the past eight years. The materials are all common, obtainable, and can be assembled by the hospital carpenter. While not in use, the accessory table can be folded and easily stored.

When the operating room is set up, the hinged boards are placed on the table and fastenings applied to the lower board. These may be either trunk straps or knee straps which are used to hold adult patients to the operating table. The patient is then placed on the working surface, which is the upper board, and fastened into position with the trunk straps. After anesthesia is established the patient is then raised into the position, which is sufficiently vertical to allow proper drainage from the mouth. This corresponds to Rose's position in which blood and mucus may flow over the incisor teeth. The anesthetist may insert a small ether hook into the mouth, or an intranasal tube, and administer the anesthetic from an attached machine. The drapes are arranged and a stitch of silk placed in the tongue and fastened to the topmost sheet, thus holding the tongue out of the field. The operator is then ready to proceed. The photographs are self-explanatory.

Specifications.—The working model of the table now in use is made of wood which has been treated with a suitable coating of white enamel. It might be possible to reproduce this table with thin sheets of stainless steel, Monel metal, or cast aluminum with some saving of weight although the table as now constructed is easily handled. Overall dimensions of the boards are 42 by 18 by $\frac{7}{8}$ inches. They are hinged together at one end with two standard hinges. The top board, or work-

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ing surface, carries the slots and straps and the hinged support on its back. The lower board which is strapped to any operating table carries the slotted angle irons which support the working surface when the rods are placed in position.

The working surface provides six slots parallel to each other, 6, 5 and 3 inches in length and all ½ inch wide. The two slots at the top of the board are ½ inch long and 2 inches wide. The lowest slots are 4 inches long and 2 inches wide. The straps used are ordinary 6 foot

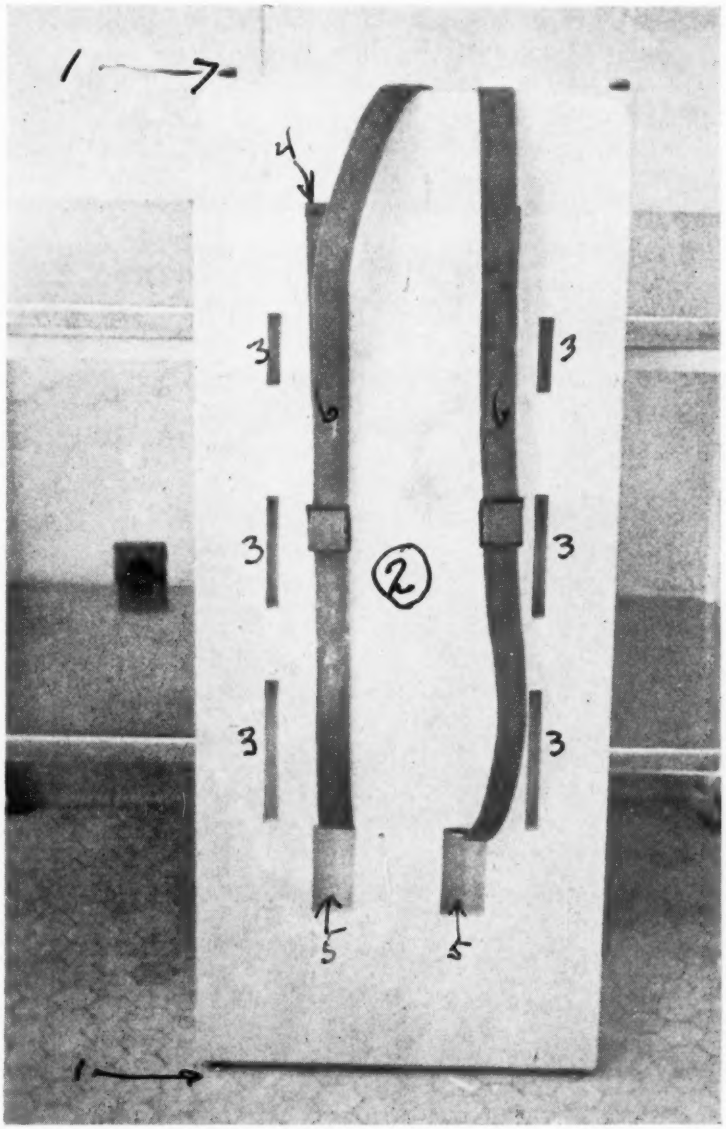

Fig. 1.—The working model stands on the floor resting against a common instrument table for the purpose of comparison of sizes. Shown here are six slots arranged in pairs (*3*) and four paired slots (*4* and *5*) arranged above and below (*3*). There are also metal gliders (*1*) and trunk straps (*6*) shown on the upper surface upon which the patient is placed (*2*).

Fig. 2.—A patient has been placed on the accessory table after it has been fixed to the lower table by two web straps (7). Note the use of the narrow slots for fixation of the patient by means of a wide band and the crossing of the trunk straps over the shoulders for use of suspension in the vertical position (8).

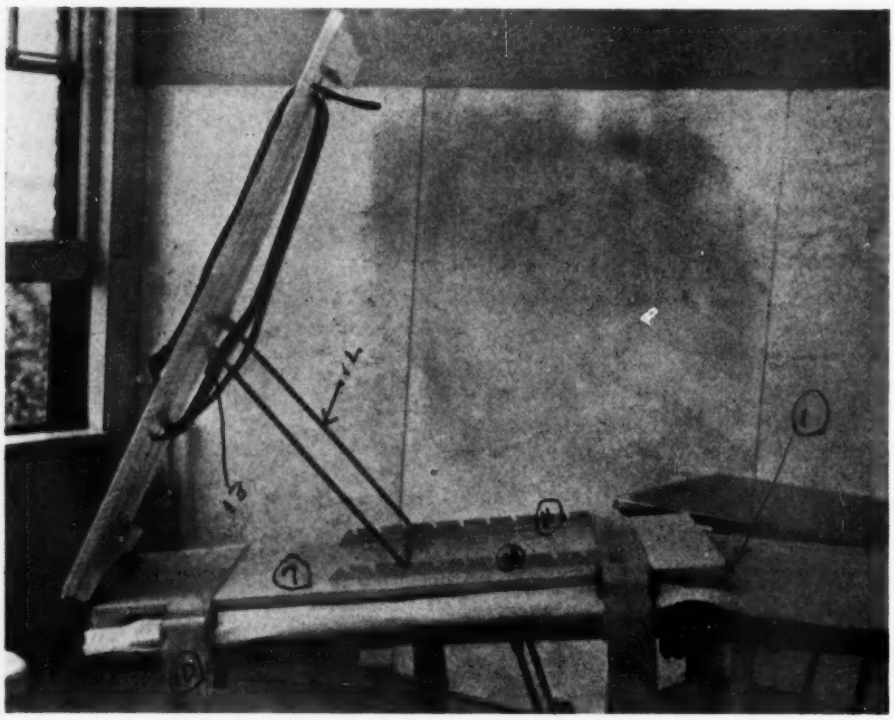

Fig. 3.—Side view of hinge (9), web knee straps (*10*) and angle iron (*11*) slotted to receive ⅝ inch U-shaped rod support (*12*). Hook-and-eye hinge (*13*) attached to rod support and table.

length, webbed trunk straps. Two gliding casters are placed on one end of each board. These should be at opposite ends. On the underside of the working surface are provided the hinges to which this board is attached to the lower board and also the hinged hook and eye and the rod support. The rod is bent so that it is U shaped. It is of ⅜ inch material and may be steel, bronze, or iron. On the lower board there is provided two parallel right angle iron bars, 21 inches long, each containing nine slots about 2 inches apart. Into these slots the U-shaped supports fit. The supporting rods are 15 inches long.

ASEPTIC APPENDECTOMY—THE APPLICATION OF THE PARKER-KERR SUTURE TO THE APPENDICAL STUMP

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THE purpose of this communication is to describe in detail the application of the Parker-Kerr suture to the appendical stump, obviating the necessity of ligating the stump. Asepsis in dividing the appendix is achieved by use of the cautery.

Since the first appendix was successfully removed, there has been considerable debate concerning the ideal appendectomy. Numerous series of appendectomies have been reported with very low mortality employing ligation and inversion of the appendical stump within a purse-string suture. Other series have been reported with similar mortality rates in which the stump was simply ligated and permitted to remain unperitonized within the abdomen. The chief criticism of either method centers about the possibility of: (1) rupture of the appendical stump after appendectomy with consequent peritonitis and abscess formation; (2) bowel obstruction from adhesions of the ileum to the cecum at the site of appendectomy; (3) mucocele formation in the stump; and (4) secondary appendicitis occurring at a later date in a stump left too long after division of the appendix.

It is felt that the method to be described successfully precludes all of these complications better than any other method. It adapts the basic principles of aseptic intestinal anastomosis to appendectomy.

METHOD

Although the actual treatment of the appendix to be described can be used with any type of incision, it will be assumed that the operation is for appendectomy alone.

For this operation, a McBurney approach to the cecum is used. The skin incision is made in a transverse-oblique direction in the line of the skin folds, over McBurney's point. After the abdomen is opened, the cecum is mobilized, and the appendix is delivered into the wound. The mesoappendix is ligated in the usual manner, with one or more ligatures, usually of No. 1 or No. 0 plain catgut. As the mesoappendix is divided between ligatures, it is peritonized with interrupted, fine, single silk sutures while the appendix remains attached to the cecum.

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The base of the appendix must be completely mobilized from all peritoneal bands connecting it to the cecum. A small straight hemostat is then placed across the base of the appendix, close to the cecum, but not including the cecum. The hemostat is set so that the handle and shaft are parallel to the ascending colon and parallel to the longitudinal tenia with the tips of the clamp pointing toward the base of the cecum

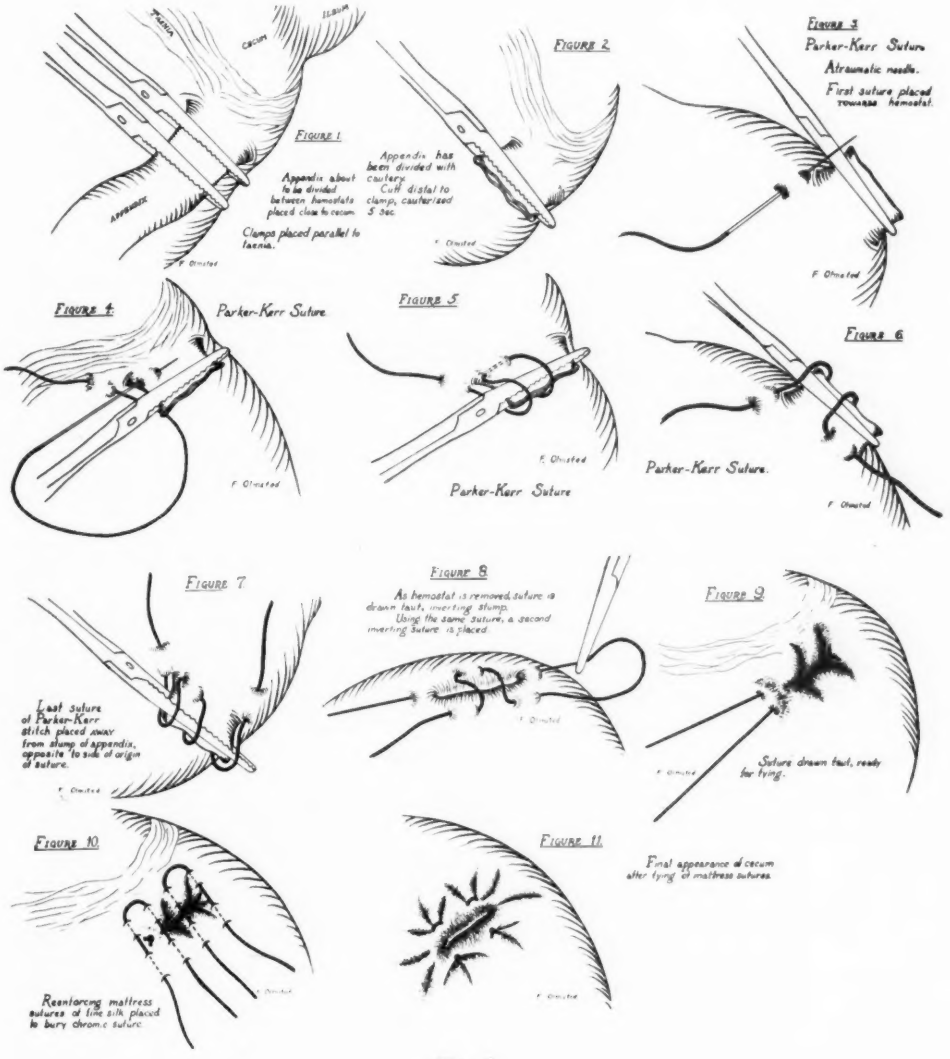
FIGURE 1
Appendix about to be divided between hemostats placed close to cecum
Clamps placed parallel to taenia.
FIGURE 2
Appendix has been divided with cautery. Cuff distal to clamp, cauterized 5 sec.
FIGURE 3
Parker-Kerr Suture
Atraumatic needle.
First suture placed toward hemostat.
FIGURE 4
Parker-Kerr Suture
FIGURE 5
Parker-Kerr Suture
FIGURE 6
Parker-Kerr Suture.
FIGURE 7
Last suture of Parker-Kerr stitch placed away from stump of appendix, opposite to side of origin of suture.
FIGURE 8
As hemostat is removed, suture is drawn taut, inverting stump.
Using the same suture, a second inverting suture is placed.
FIGURE 9
Suture drawn taut, ready for tying.
FIGURE 10
Reenforcing mattress sutures of fine silk placed to bury chromic suture.
FIGURE 11
Final appearance of cecum after tying of mattress sutures.

Fig. 1.

(Fig. 1). A second hemostat is set distal to the first. The appendix is divided with the actual cautery so that a cuff of appendix about 1 mm. long is left distal to the proximal clamp (Fig. 2). This cuff is further cauterized for five seconds. A Parker-Kerr suture of No. 00 chromic catgut on a weld-on needle is then placed across the clamp holding the base of the appendix (Figs. 3 to 7).

The clamp is withdrawn as the suture is tightened, inverting the stump of the appendix into the cecal lumen. With the same chromic suture, a second continuous suture is placed over the Parker-Kerr suture line (Fig. 8). The suture is then tied at the same place where it was begun (Fig. 9). Two or three mattress sutures of single intestinal silk are placed so as to cover the knot of the catgut suture and to re-enforce the remainder of the chromic suture line (Figs. 10 and 11).

COMMENT

Several steps of this procedure are worthy of emphasis. Care must be taken to free the appendix from the cecal wall so that the Parker-Kerr suture can be accurately placed submucosally. The hemostats applied to the appendix must be placed parallel with the longitudinal tenia so that the suture line will not encroach upon the ileocecal valve. Cauterization of the short cuff distal to the clamp holding the base of the appendix insures hemostasis and temporary sealing of the stump. The initial and final placing of the Parker-Kerr suture must be as indicated (Figs. 3 and 7) so that proper inversion of the ends of the stump will occur. The Parker-Kerr suture line must be re-enforced with interrupted nonabsorbable sutures.

CONCLUSION

The advantages of this method are: (1) Aseptic division of the appendix is achieved with the actual cautery. (2) The appendix is divided at the cecum so that mucocele or appendicitis of the stump is impossible. (3) The stump of the appendix is inverted into the cecal lumen without ligation so that no blind pocket between the stump and the inverting suture is possible. (4) No unperitonized surface of the cecum is present.

Fifteen consecutive appendectomies have been performed in this fashion without complication.

LESIONS OF THE TERMINAL COLON ASSOCIATED WITH URINARY DISTURBANCES

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IT IS obvious that patients with urinary symptoms eventually fall into the hands of urologists for diagnosis and treatment. This being the case, it naturally follows that urologists must constantly be on the alert to the ever-present possibility that many symptoms which, on the surface, appear to be due to diseases involving the genitourinary tract may actually be due to extraneous conditions. It is the purpose of this communication to deal with lesions of the terminal bowel which give rise to urinary disturbances.

Hemorrhoids.—Hemorrhoids constitute one of the more frequent conditions associated with prostatism, especially when prostatism is due to prostatic hypertrophy. In these cases hemorrhoids result from pressure exerted upon the hemorrhoidal plexus of veins by the enlarged gland, and from straining during urination. Patients frequently seek relief of the annoying hemorrhoids rather than of the prostatic condition to which they owe their origin. After ascertaining the absence of a lesion in the rectum or rectosigmoid responsible for the hemorrhoids, it is necessary to make careful inquiries regarding the exact status of the urinary tract. In older men special inquiries should be made to establish the condition of the prostate gland. Once it is determined that the patient has an obstructive prostate, one may be reasonably certain that measures directed to its removal will, in the vast majority of instances, lead to spontaneous amelioration or complete disappearance of the hemorrhoids. On the other hand, surgical intervention for the relief of hemorrhoids without removal of the offending prostate is certain to lead to recurrence of symptoms.

Carcinoma of the Rectum.—One of the most frequent sources of error in urologic diagnosis is the tendency to overlook the presence of carcinoma of the rectum, particularly among men with hypertrophy of the prostate gland. This is especially true in patients with symptoms of prostatism who, on perfunctory digital rectal examination, present grossly enlarged prostates. It is extremely tempting in such instances to indulge in a snap diagnosis of prostatic hypertrophy with its implication that the enlarged gland is the underlying and sole cause of the urinary disturbance. It must be realized, however, that the discovery of an enlarged prostate on rectal examination does not necessarily imply that the prostate is the cause of the prostatism. It is not unusual to see patients who, although disclosing enlarged prostates on

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rectal digital examination, fail to show any evidence of intrusion of prostatic lobes into the urethra, or evidence of vesical neck distortion due to the enlarged prostates on cysto-urethroscopy. Visual disclosures of this character signify that the prostate plays no pertinent role in the causation of presenting genitourinary symptoms.

Urologists can readily attest to the fact that certain lesions in juxtaposition to the vesical neck, although extrinsic to the genital tract, can and do give rise to prostatism. The most frequent among such lesions is carcinoma of the rectum. It is also common knowledge that men harboring grossly enlarged prostates are prone to present symptoms referable to the lower bowel, especially the rectum. The more common symptoms are constipation, a sense of fullness in the rectum, hemorrhoids, and rectal bleeding. The collateral symptoms can readily be explained on the ground of pressure exerted by the enlarged prostate upon the rectum itself and upon the hemorrhoidal plexus of veins and nerves.

While loss of weight would tend to make one alert to the possibility of some malignant lesion, it is not at all unusual to see long-standing cases of prostatism, especially among elderly patients who have lost weight and strength solely because of an associated chronic azotemia. This same explanation holds true for secondary anemia which is frequently encountered in both conditions.

In order to avoid the danger of overlooking the presence of carcinoma of the rectum in patients presenting clear-cut histories of prostatism, it is necessary to establish definite and fixed rules in examining these patients. Of primary consideration is a carefully taken history, with special emphasis placed upon changes in bowel habits, such as constipation or diarrhea. Rectal bleeding is an extremely important symptom, even in patients suffering from large hemorrhoids.

Every prostatic should have a careful digital rectal examination instead of the perfunctory one usually performed in palpating the prostate. In view of the fact that in the vast majority of cases rectal carcinomas are situated within reach of the examining finger, and in view of the fact that in men with pronounced enlargement of the prostate it is at times difficult to examine accurately the region directly above the base of the prostate by means of the usual technique, it becomes necessary to employ methods designed to render maximum information. I have found that in order to accomplish this end, patients should be examined while lying in the supine position, with knees drawn up to the chin. The right index finger is introduced into the rectum, and the palm of the left hand exerts gentle downward pressure over the suprapubic region, while the patient is instructed to bear down. The examining finger is then carefully swept around the entire circumference of the rectum. Through this maneuver the examiner is enabled to extend the range of palpation considerably above the base

of the prostate, regardless of its size. Detection of anything suspicious warrants immediate proctoscopic examination to visualize the lesion and to obtain specimens for biopsy. Failure to palpate anything suspicious does not relieve the examiner of the responsibility of performing proctosigmoidoscopic examinations upon patients presenting evidence of bowel disturbances, regardless of how slight they might be.

Having detected the presence of a carcinoma of the rectum, it becomes necessary to ascertain its role in the causation of urinary symptoms. Careful cysto-urethroscopic examination should, in most instances, readily establish the role played by the enlarged prostate. An enlarged prostate gives rise to prostatism by interfering with the free flow of urine from the bladder by virtue of its tendency to compromise the vesical neck or prostatic urethra, or both. Regardless of its size, as determined by rectal digital examination, failure of the gland to encroach upon the prostatic urethra or vesical neck, in my opinion, definitely rules out the possibility of the prostate as playing any role in the causation of urinary symptoms. A rectal carcinoma which gives rise to urinary disturbances usually presents certain fairly characteristic cystoscopic features. The most common is a localized area of bullous edema involving a portion of the base or posterior wall of the bladder, while the remainder of the bladder wall appears relatively normal. This finding suggests a bowel lesion which has become adherent to the bladder through inflammatory adhesions or as a result of actual invasion. Occasionally one may even see, in long-standing cases, an actual communication between the bladder and rectum (rectovesical fistula). In very early cases, instead of an area of bullous edema, one may note a patch of hyperemia involving a localized area of bladder wall. A not unusual finding is a localized area of invagination of a portion of the posterior bladder wall into the vesical lumen, with no apparent change of the mucosa covering this portion of bladder.

Another problem which is occasionally encountered is a patient who presents an enlarged prostate on rectal digital examination along with a rectal carcinoma. Cystoscopy, however, discloses a well-defined median bar, but no intrusion of the prostate into the bladder or prostatic urethra.

The therapeutic problem involved in this group of cases is naturally dependent upon findings. When the prostate is enlarged and obstructing, treatment must obviously be directed to the removal of the gland. While transurethral resection is the procedure of choice, enucleation by the perineal or suprapubic routes is far more preferable under certain circumstances, particularly when associated with renal insufficiency. Following relief of the prostatic obstruction, appropriate therapy should be instituted to remove the rectal lesion as quickly as is compatible with safety. However, should cystoscopy fail to disclose any causal relationship between the physical characteristics of the prostate and the presenting urinary symptoms, surgical intervention

should be directed to the eradication of the rectal carcinoma without giving any consideration to the gland. Urinary symptoms in this group of cases usually disappear spontaneously following the removal of the rectal carcinoma.

Lesions of the Sigmoid and Rectosigmoid.—

Diverticulitis of the Sigmoid: Acute or chronic diverticulitis of the sigmoid frequently results in adhesions binding the affected segment of the sigmoid to a portion of the posterior wall of the bladder. This condition inevitably results in urinary symptoms closely simulating prostatism. In fact, the urinary symptoms may be so prominent as to overshadow completely any symptoms referable to the colon, so that in many instances patients will consult the urologist rather than the intestinal surgeon for relief of symptoms. Careful cystoscopic study of the bladder usually discloses a localized area of congestion or bullous edema of the posterior vesical wall, and in severe and long-standing cases may reveal the opening of a sinus tract connecting both viscera. Such lesions often simulate neoplasms so closely that it is difficult to differentiate between the two conditions without the aid of biopsies. Detection of this condition, in the course of a routine cystoscopic study warrants careful interrogation of the patient regarding bowel habits. As usually happens, patients falling within this group are stocky in build and present histories of long-standing episodes characterized by pain in the left loin associated with constipation or diarrhea, with or without bouts of fever. While passage of blood in the stool may be present, it is not as commonly encountered in cases of diverticulitis as in cases of carcinoma of the bowel. Hematuria is not an unusual symptom. Correct diagnosis can always be established by means of proctoscopy and double contrast barium enemas.

Although acute diverticulitis often subsides under appropriate conservative medical care, it is very doubtful whether anything short of surgery will suffice to bring relief in cases associated with adhesions between the involved colon and the vesical wall. In such cases the logical procedure is to set the involved segment of bowel completely at rest by means of a "defunctioning" colostomy placed at some distance proximal to the sigmoid, preferably in the transverse colon. A colostomy of this type, in order to be effective, should be permitted to function for from nine to twelve months. If, at the expiration of this time, it is determined by x-ray and sigmoidoscopy that the process is still active, the affected segment of bowel should be extirpated and the continuity of the bowel restored. If it has been ascertained that in addition to the diverticulitis, an obstructing prostate is also present, measures should be taken to remove the prostate, perferably by transurethral resection whenever practical. Prostatectomy should be performed prior to the establishment of colostomy when the obstruction is marked. In

the milder types of obstruction, it is better to wait one or two months after the affected segment of bowel has been defunctioned, before resorting to prostatectomy.

Carcinoma of the Sigmoid and Rectosigmoid: Malignant tumors involving the sigmoid and rectosigmoid give rise to urinary symptoms as a result of pressure upon the bladder, or of direct involvement of the vesical wall by carcinomatous infiltration, or by inflammation. Pressure manifestations usually occur in cases where the tumor is of fairly large size and located in the mobile portion of a redundant sigmoid flexure, in which case the tumor drops down deeply into the pelvis between the rectum and bladder. In these cases cystoscopy reveals an invagination of a segment of posterior bladder wall into the vesical lumen without any evident change in the mucosa over the site of the intrusion.

Urinary symptoms are elicited more frequently in cases where tumors have become adherent to segments of bladder wall by carcinomatous infiltration or by inflammatory adhesions. Such tumors are more apt to be found in the lower third of the rectosigmoid. It is not an infrequent occurrence to find an abscess present in the depth of the pelvis due to an old perforation of the bowel at the site of the tumor. When this occurs, vesical symptoms are often well pronounced.

Since patients falling within this group are more likely to consult the urologist before the abdominal surgeon, it is necessary for him to be constantly on the alert if he is to avoid grave errors in diagnosis. It is well known that tumors involving the left half of the colon are notorious for their tendency to occlude the bowel. Because of this fact, it is imperative to interrogate patients carefully concerning changes in bowel habits, regardless of the fact that the primary complaint may apparently be concerned with the urogenital tract. Since lesions in this segment of bowel are also prone to bleed, a history of bleeding from the rectum assumes added significance, and should never be passed over lightly as being due to hemorrhoids which frequently accompany prostatism. Excessive loss of weight, especially when accompanied by asthenia, is suggestive of this type of lesion, particularly in the absence of azotemia or carcinomatous changes within the prostate gland. The merest suspicion of malignancy warrants prompt sigmoidoscopy and barium enema studies to clinch the diagnosis.

The same principles of therapy apply here as in the conditions previously discussed. When the intestinal lesion is the sole cause of urinary disturbances, the indicated procedure is to eradicate the lesion in the bowel. If, however, lesions involving both the colon and the bladder or prostate are present, it is my belief, that the latter should have priority; and the treatment should be so designed as to provide prompt relief of the urinary conditions, with the least manipulative efforts. Suprapubic cystostomy should always be performed where actual or

threatened perforation or fistula formation between the colon and bladder exists. This procedure need in no way interfere with the surgical approach to the intestinal tumor. In fact, suprapubic cystostomy is often of inestimable value in the surgical management of cases of intestinal carcinomas, particularly when lesions are situated low in the bowel and require considerable manipulation of the bladder and its sympathetic nerve supply in the course of the operative procedure.

CASE REPORTS

CASE 1.—M. P., aged 47 years, was seen June 25, 1940, complaining of increased diurnal and nocturnal urinary frequency, dribbling, and difficulty in starting the stream, associated with belching and pyrosis. Fifteen years before he had had a gastrectomy performed for ulcer. Rectal examination disclosed internal hemorrhoids, fissure, and fistula in ano. Following hemorrhoidectomy along with the excision of the fissure and fistula, he made an uneventful recovery with complete disappearance of all urinary symptoms.

CASE 2.—J. B., aged 61 years, seen Sept. 23, 1930, complained of increased diurnal and nocturnal urinary frequency, dribbling, pain on defecation, and bleeding from the rectum. Proctoscopic examination disclosed a fissure in ano with hemorrhoids, for which surgery was performed. Following this he made an uneventful recovery, with complete disappearance of all urinary symptoms.

Comment.—Cases 1 and 2 both illustrate prostatism due entirely to spasm of the muscles about the vesical neck which resulted from irritative lesions of the rectum and anus. Appropriate treatment directed to these causes led to complete relief of symptoms.

CASE 3.—J. D., aged 60 years, was seen Feb. 2, 1943, complaining of retention of urine, dribbling, and frequency of urination, of several weeks' duration. There was occasional constipation, but no bleeding from the rectum.

Investigation of the genitourinary tract revealed an enormous enlargement of the prostate. Kidneys were normal.

Physical examination.—Blood pressure was 140/90; heart and lungs, normal. There were large thrombosed hemorrhoids. By rectum the prostate felt enormously enlarged and irregular.

Following a two-stage suprapubic prostatectomy, all symptoms including those due to the hemorrhoids disappeared.

CASE 4.—J. H., aged 66 years, was seen Aug. 18, 1943, complaining of prostatism and lack of appetite with belching, abdominal distention, and constipation of a few years' duration.

Diagnosis.—Median bar, chronic cholecystitis, and internal hemorrhoids. Cholecystectomy and appendectomy were performed. During convalescence he suffered an attack of prolapsed thrombosed hemorrhoids, with pain and bleeding on defecation. Following transurethral resection urinary symptoms disappeared, and the hemorrhoids receded.

Comment.—Cases 3 and 4 are examples of hemorrhoids associated with prostatic disease which subsided spontaneously following prostatectomy.

CASE 5.—S. R., aged 64 years, was admitted to the hospital on the night of April 28, 1941, with an attack of acute urinary retention. He stated that for

the past few months he was troubled with diurnal and nocturnal urinary frequency. During this period he had lost twenty-five pounds in weight. Only after careful questioning was it ascertained that he had occasional bouts of diarrhea alternating with periods of constipation.

Physical examination disclosed a thin, cachectic, chronically ill patient who presented no outstanding signs. Blood pressure was 170/90. The prostate, as felt per rectum, did not appear particularly enlarged or hard.

Cystoscopy disclosed marked contracture of the vesical neck, with a well-defined median bar. The lateral lobes were not enlarged. On the anterior wall of the bladder near the vault there was a small area where the mucosa appeared reddened and edematous. This finding led us to believe that we might be dealing with a contact inflammation resulting from some intra-abdominal lesion lying adjacent to the bladder. It was fairly certain that the genitourinary findings did not fully explain the presenting clinical picture, particularly the general cachectic appearance of the patient, and the complete urinary retention.

Sigmoidoscopy disclosed a tumor involving the anterior wall of the rectum, situated 4.5 inches from the anus. Biopsy showed adenocarcinoma of the rectum.

Exploratory laparotomy, performed May 13, 1941, revealed a large mass about the size of an orange involving the sigmoid flexure, upper rectum, one loop of small intestine, and the posterior vesical wall. A metastatic nodule was present in the right lobe of the liver. A loop colostomy was performed above the tumor. Following this, all urinary symptoms disappeared, indicating thereby that the urinary retention was primarily due to spasm rather than to mechanical obstruction of the vesical neck by the prostate gland.

CASE 6.—S. McI., aged 59 years, was first seen July 14, 1930, complaining of increased diurnal and nocturnal urinary frequency, dribbling, difficulty in starting the stream, and pyuria, of eight months' duration.

The physical examination was essentially negative. On rectal examination the prostate felt small and elastic.

Cystoscopy disclosed a thickened, trabeculated bladder with a well-defined median bar and contracture of the vesical neck. The lateral lobes of the prostate were not involved.

July 29, 1930, the bar was excised with the Collings electrotome, and the patient was discharged from the hospital August 1. He returned Oct. 3, 1930, and stated that since leaving the hospital he had noticed rectal irritability which had become more pronounced during the preceding five days. Two months previously he had noted blood in the stool, which persisted for two days but did not recur. There was no diarrhea, constipation, or loss of weight. Digital rectal examination revealed a hard, annular carcinoma involving most of the anterior wall of the rectum, just barely palpable to the examining finger.

The symptoms of prostatism presented by this patient were undoubtedly due to spasm induced by the carcinoma of the rectum, which had been overlooked at the time of his first examination.

CASE 7.—H. K., aged 58 years, was seen May 7, 1928, complaining of difficulty in urinating, frequency of urination, and dysuria. On rectal examination an ulcerating carcinoma was found involving the lower anterior wall of the rectum. The prostate was not enlarged.

CASE 8.—A. F., aged 59 years, was seen June 18, 1943, complaining of pain in the left lower abdomen, bleeding from the rectum, painful defecation, increasing constipation, and frequency of urination, voiding six times during the day and one to two times at night, and slight dysuria.

Physical examination.—Blood pressure was 140/90. There was tenderness of the left lower abdomen. Proctoscopy revealed bleeding internal hemorrhoids. Sigmoidoscopy was negative. Barium enema showed a well-filled colon, with no evident defect.

The patient was seen at the office on several occasions where the hemorrhoids were touched up with silver nitrate. Pain persisted, however, and operation was advised and accepted.

At operation an infiltrating carcinoma of the sigmoid flexure was found, consisting of a mass about the size of an orange infiltrating into the bladder.

CASE 9.—J. B., aged 53 years, was seen Oct. 14, 1943, complaining of right renal colic of one week's duration, frequency of urination, voiding six times during the day and two to three times at night, pain in the right lower abdomen, and dull right lumbar pain. Stools were normal; there was no constipation.

Physical examination.—Blood pressure was 130/80. There was right costovertebral tenderness and tenderness and rigidity over the entire abdomen. Rectal examination was negative, rectal temperature 103.2° F.; pulse, 100.

He was immediately hospitalized and prepared for operation, at which time a ruptured tumor of the sigmoid and generalized peritonitis were found. A Mikulicz resection of the bowel was performed. Pathologic diagnosis was diverticulosis of sigmoid; acute purulent diverticulitis of sigmoid with intramural abscess formation and fibrinopurulent perisigmoiditis; appendix showing mild acute serosal inflammation.

CASE 10.—M. N., aged 52 years, was seen April 11, 1943, complaining of pain in the epigastrium and left lower quadrant on and off for several weeks, bleeding from rectum, occasional attacks of diarrhea alternating with constipation, loss of weight, frequency of urination, and occasional dysuria.

Physical examination revealed a well-developed man, who appeared ill. Blood pressure was 130/90. There was tenderness present in the left iliac fossa. Rectal examination was negative.

Fluoroscopy of the colon with barium enema showed the column of barium reaching up to the region of the rectosigmoid, beyond which no barium could be seen. The patient complained of a great deal of distress, making it impossible to allow more barium to enter the bowel.

X-rays of the colon showed the ampulla very much dilated. There was a jagged deformity of the lower rectosigmoid, with several diverticula.

Sigmoidoscopy failed to reveal any lesion as far as the sigmoidoscope could be introduced, which was a distance of 7 inches.

Blood Count.—Hemoglobin, 108 per cent; red blood cells 5,760,000; white blood cells, 16,400; polymorphonuclears, 70 per cent; lymphocytes, 16 per cent; rods, 6 per cent; metamyelocytes, 1 per cent; monocytes, 7 per cent.

Urinalysis.—Specific gravity, 1.021; pH, 5.7; albumin and sugar, negative; microscopic occasional white blood cells; few epithelial cells and mucus.

The patient was admitted to the hospital on the morning of April 13, 1943, complaining of pain in the lower abdomen, and temperature of 102° F., and presenting signs of peritonitis. He was prepared for immediate laparotomy.

At operation the rectosigmoid just above the peritoneal reflexion presented a large mass (diverticulum) about the size of an adult fist adherent to the promontory of the sacrum behind, extending over to the right of the midline and adherent to the right iliac fossa and to the bladder in front. The right lateral margin of this mass was covered with fibromucopurulent exudate. There was an escape of free pus from the peritoneal cavity. A double-barrel colostomy was performed.

The patient made an uneventful recovery, and was discharged from the hospital, May 9, 1943, with a well-functioning colostomy. All urinary symptoms had disappeared.

CASE 11.—M. S., aged 46 years, was seen Oct. 18, 1938, complaining of abdominal cramps following defecation, ribbonlike stools, occasional nausea and vomiting, and frequency of urination, voiding seven to eight times during the day and once at night.

Physical examination.—Blood pressure was 130/80. There was a definite tender mass situated in the left iliac fossa, which was fairly well fixed. Rectal examination was negative.

Sigmoidoscopy was negative.

Barium enema x-rays revealed marked spasticity of the sigmoid colon, with pocketing just above the sigmoidal loop. After evacuation, barium remained behind in this loop.

The patient was sent to the hospital where a "defunctioning" colostomy was performed for diverticulitis of the sigmoid flexure. Convalescence was uneventful, and he was discharged nineteen days later. All urinary symptoms had disappeared.

He was seen at the office two months later, when a barium enema revealed no evident defect in the colon, as previously noted. There was no recurrence of urinary disturbance. April 27, 1939, he was again sent to the hospital where the colostomy was closed.

SUMMARY AND CONCLUSIONS

This communication deals with certain lesions of the terminal colon which give rise to urinary symptoms suggesting involvement of the urogenital tract. It emphasizes the fact that prostatism may be due solely to involvement of this segment of the bowel or in combination with obstructive hypertrophic prostates. When prostatism is primarily the result of lesions in the terminal colon it can be attributed to spasm of the vesical neck resulting from irritation of the anterior rectal wall. Histories taken from patients presenting urinary symptoms must include precise and careful interrogation regarding bowel habits. Carefully performed rectal examination is imperative in all urologic patients, especially prostatics. Whenever symptoms suggest the possibility of rectal or anal involvement, digital examination should be supplemented by proctoscopic and x-ray studies of the colon. In the presence of symptoms suggesting colonic irritation, when preoperative examinations have failed to reveal evidence of colonic pathology, it is essential to perform careful rectal examination following the removal of the obstructing prostate. Appropriate treatment of an obstructing prostate should take precedence unless intestinal symptoms strongly predominate. A series of cases is presented to illustrate the various phases of this problem.

A NEW TECHNIQUE FOR REPAIR OF FACIAL PARALYSIS WITH TANTALUM WIRE

WILLARD W. SCHUESSLER, M.D.,* WASHINGTON, D. C.

IT HAS been suggested by J. C. White† that tantalum wire be used for repair of facial paralysis, rather than fascial strips. A new technique has been devised at The Walter Reed General Hospital for the treatment of facial paralysis.

The method is simple and effective, and stabilizes the mouth, lower end of nose, and eyelids, thereby transmitting some elements of expression to the paralyzed side. There are certain advantages when this technique is employed:

1. The operation is performed under local anesthesia.
2. No disability or bed rest is necessary for the patient, as no incision is made for the removal of fascia from the thigh.
3. When the wires are in position, the patient can sit up and the wires can be tightened to the proper tension, thereby doing away with the amount of sagging in the paralyzed side which occurs when patient is lying down.
4. There is no swelling or hemorrhage in the tissues of the paralyzed side because of the very slight trauma which is produced by the small caliber of the local needle.
5. This method can also be used to hold the muscles in balance during the time the nerve is regenerating, and when fully recovered the wires can be removed.

TECHNIQUE

Only tantalum wire .007 gauge, long local needle 18 gauge, and clips made from tantalum foil .025 thick are used. The clips are cut one-half inch long, one-fourth inch wide, and have a hole punched in each end; the square corners are rounded and then folded onto themselves over the tip of a small mosquito forceps (Fig. 1).

The temporal region is shaved and thoroughly prepared. A hockey stick incision about one and one-half inches long is made above the zygoma over the temporal muscle in the hair-bearing area, in order to hide the scar in the hair. The temporal fascia is exposed. A long curved 18 gauge needle is then inserted into the incision, passing downward between skin and mucosa, injecting novocain ½ per cent to just lateral to the midline on the normal side of the upper lip. The needle is then brought close to the surface and a small wheal of local anesthesia

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Fig. 1.—Tantalum foil molded on mosquito forceps to be placed around muscle to prevent wire from cutting through the muscle.

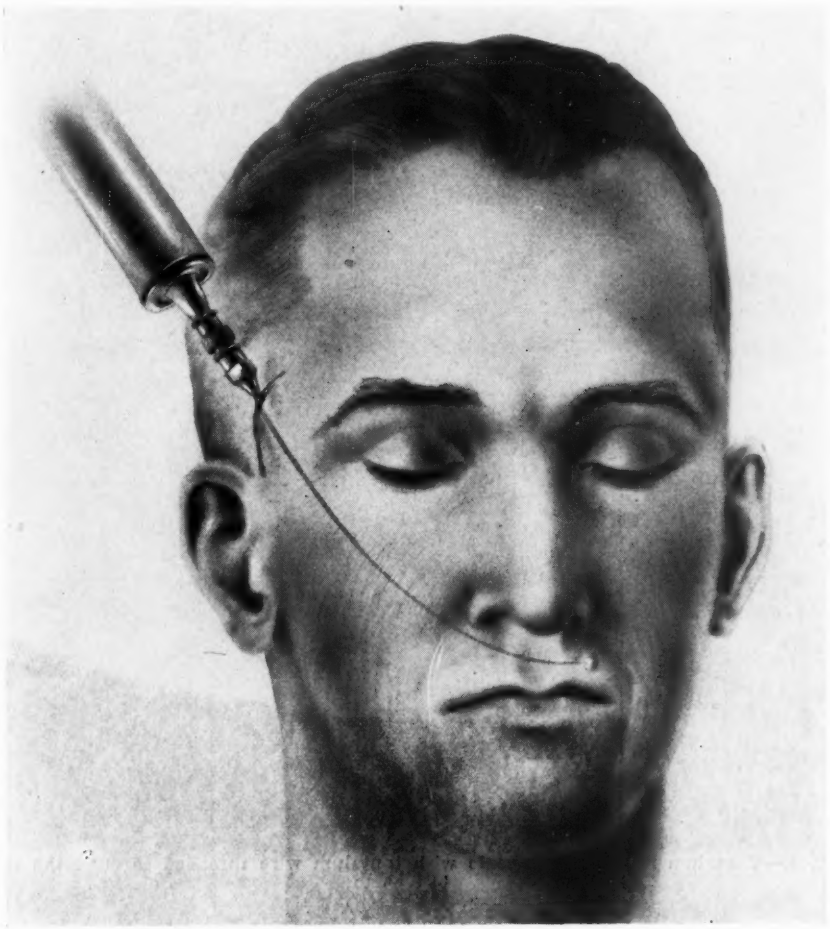


Fig. 2.—Drawing shows incision and position of fine gauge needle.

is then made. A stab incision is then made through the wheal and a bundle of muscle is tunneled by use of small scissors and mosquito forceps. The previously described clamp is then placed around the

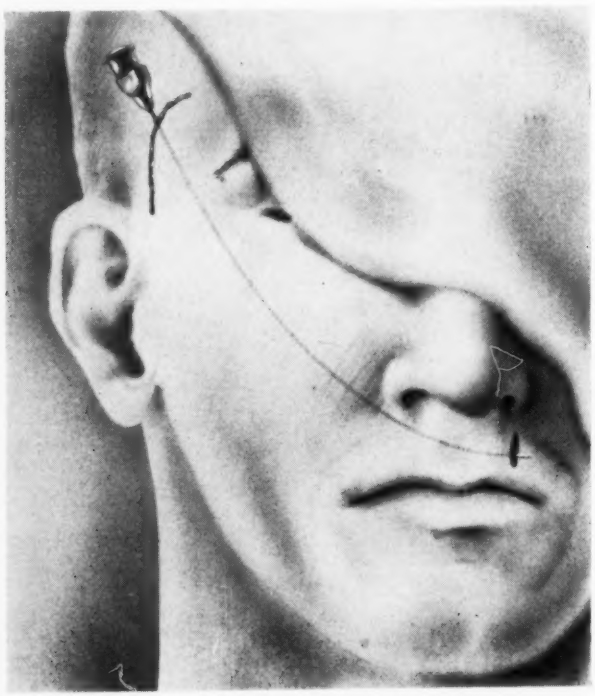


Fig. 3.—Position of incision over tip of needle.

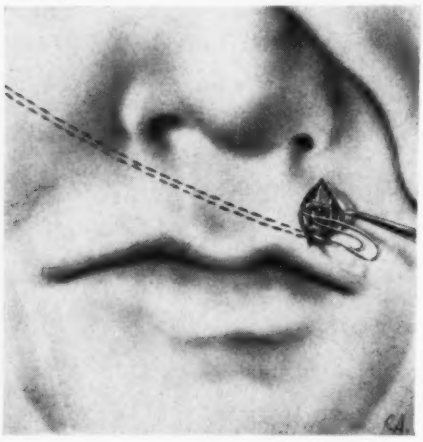


Fig. 4.—Tantalum foil around muscle with tantalum wire threaded through the local needle.

muscle bundle and the tantalum wire is passed through the holes in the clamps; the syringe is removed, the ends of the tantalum wire are passed through the bore of the local needle to the incision in the tem-

poral area, and the needle is removed. The incision in the lip is closed with fine silk or horsehair (Figs. 2, 3, and 4).

The same procedure is carried out for the angle of the mouth on the paralyzed side, and also just lateral to the midline of the normal side of the lower lip (Fig. 5).

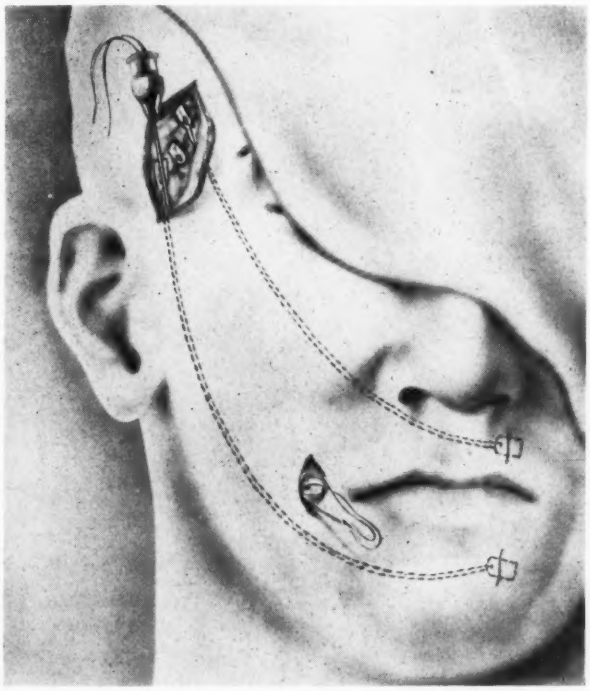


Fig. 5.—Wires in position.

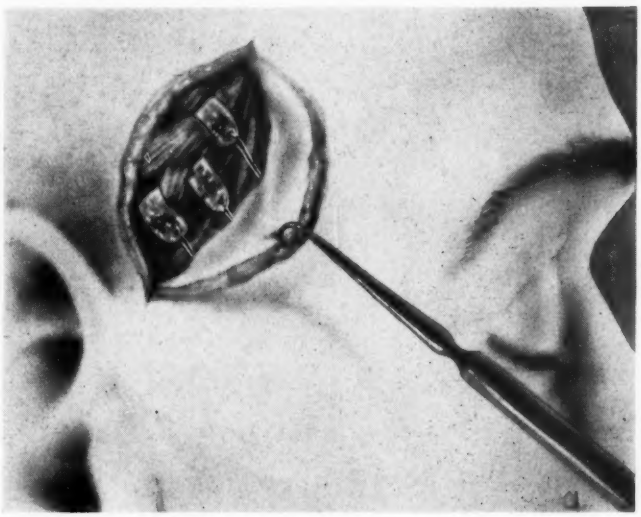
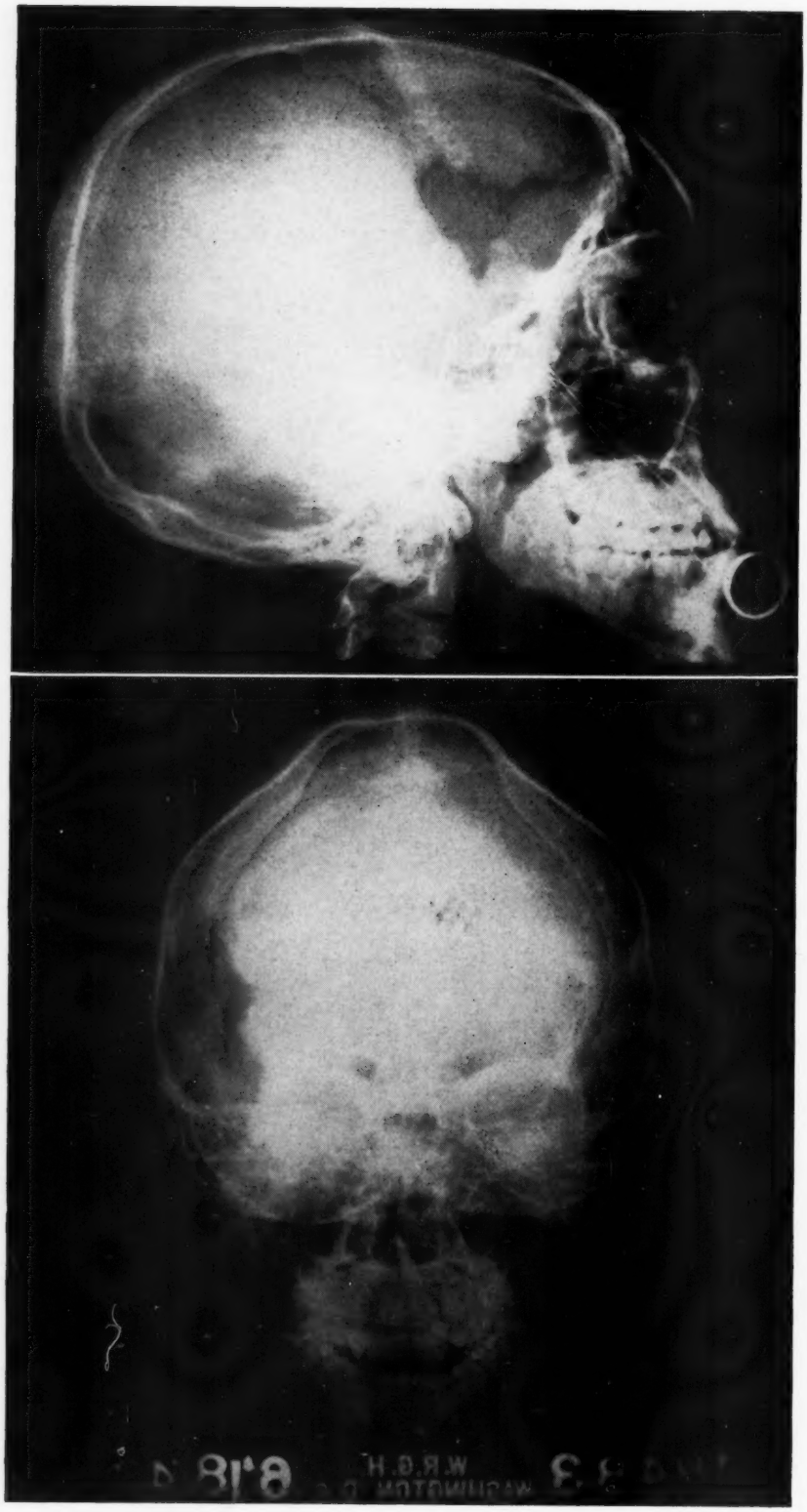


Fig. 6.—Drawing of wires with protective tantalum foil attached to temporal muscle and fascia.



Figs. 7 and 8.—X-rays demonstrating position of wires.

Small parallel incisions are made in the temporal fascia and muscle and small bundles are thus formed from the fascia and muscle to which the small tantalum clamps are fastened. The tantalum wire which has passed through the local needle into the temporal incision is reinserted

Figs. 9 and 10.—Appearance of patient before and after operation.

into the small holes of the clamps. The patient is placed in a setting position and the wires tightened the required amount. The excess is cut with scissors and the end is pressed down into one of the small

parallel incisions, whereby the large primary incision is closed with fine silk or horsehair. A small sterile bandage is placed over each incision (Fig. 6).

The patient can talk, eat, and walk immediately because the large pressure bandage ordinarily employed with this procedure is eliminated. Tube feeding for a period of one week, as required when fascial strips are used, is not necessary with this technique. There is very little danger of damaging the parotid duct. The possibility of perforating the oral cavity is minimized because novocain is injected ahead of the needle.

In the case described, it was not felt necessary to anchor the muscle in the nasolabial fold or the upper or lower eyelids. However, the procedure can be carried out as previously described for the mouth (Figs. 7, 8, 9 and 10).

In this procedure it would not be necessary to remove any of the superior border of the malar bone for the passage of the tantalum wire around the upper and lower eyelids for decreasing the size of the palpebral fissure and protecting the cornea.

In cases where there is an excess of sagging skin, this can be corrected by standard plastic procedures not discussed in this communication.

THE ASSOCIATION OF THROMBOPHLEBITIS WITH HEMATOGENOUS BONE INFECTION

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IN SEPTEMBER, 1941, I reported in SURGERY a case of acute hematogenous osteomyelitis of the upper end of the tibia with bacteriemia, in which the inflammatory process spread outward from the bone to involve the adjacent saphenous vein in a thrombophlebitic process, and in which control of the bacteriemia only took place after operative excision of the involved vein. At that time, I pointed out the similarity of this case to the ordinary case of mastoiditis with jugular thrombosis.

In spite of the fact that this association had occurred to me many times previously as a theoretical possibility, this was the first objective demonstration of this occurrence that I had encountered. A second similar experience has since occurred which I report herewith.

In March, 1931, the patient, then a young unmarried woman, developed a painful swelling of the middle portion of the left tibia. A biopsy showed a hemorrhagic mass in the interior of the tibia which was reported "subacute inflammation." The x-ray examination showed a cavity in the medulla of the bone. The cavity was curetted out. The final diagnosis on discharge was "an inflamed bone cyst of the tibia."

In September, 1936, there was a recurrence of pain in the same area. The lesion in the tbia was again explored operatively. There were similar findings and the wound healed uneventfully.

During the succeeding several years, there were a number of exacerbations of pain and swelling in the same bone which subsided spontaneously.

In May, 1943, the patient developed a dento-alveolar infection which suppurated and was incised and drained. Almost immediately thereafter, there was another recurrence of the previous inflammatory process in the tibia. There were no chills or excessive fever or other signs of a general infection, and the condition seemed localized to the tibial area.

This time the inflammatory swelling seemed to spread outward from the tibia into the soft parts on the anterior aspect of the leg. The patient was treated with radiotherapy. As the swelling began to subside, one could distinguish very distinctly a thrombosed vein lying in the subcutaneous tissue fairly close to the anterior border of the tibia. Under continued radiotherapy, the entire condition cleared up until final healing.

It seems likely that this was a hematogenous infection from the site of the dento-alveolar infection. The association with a phlebitis in the anatomically associated vein is again striking and adds another proof to the mechanism of hematogenous bone infection, that is, osteomyelitis.

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SIMPLE TECHNIQUE FOR MULTIPLE VENIPUNCTURES

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REPEATED venipunctures performed to obtain blood specimens over a period of several hours have long been a source of discomfort to the patient and often a difficult task for the house officer. The latter is particularly true in obese patients or those with few veins.

These difficulties can be easily surmounted by the use of an ordinary spinal puncture needle or bone marrow needle. The virtue of these types is the stylet, the end of which presents itself flush or flat with the beveled point of the needle, thereby eliminating the possibility of blood clotting in the lumen.

The most convenient site of introduction of the needle is a vein on the volar surface of the forearm. When inserted, it can be taped in place and does not impede motion of the arm during the test. If there are no favorable veins in this area, the antecubital site is used but this necessitates immobilization of the elbow or complete cooperation of the patient, which is easily obtained.

The needle may be inserted with the stylet in place, as in a spinal puncture, or usually it is placed on the syringe and the typical venipuncture performed. After the first blood specimen is obtained, the stylet is reinserted until time for the ensuing one. The stylet, when removed, either can be held by an assistant or placed upon sterile gauze. This procedure is repeated until all specimens are obtained.

The circulation of blood past the needle tip is adequate and normal, thereby assuring accuracy. Most satisfactory results have been obtained in a research project using this technique in Smith's kidney clearance tests.

It is probably not a good idea to inject dye, such as bromsulfalein, because of possibility of contamination of the needle lumen and the immediately adjacent vein wall, although perhaps this source of error could be precluded by washing the system with from 5 to 10 c.c. of saline solution after administration of the dye.

This procedure has proved efficacious in the execution of such clinical pathologic tests as bromsulfalein, glucose, and galactose tolerance tests and many others requiring several blood specimens.

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THE OPERATIVE ATTACK ON ORGANIC PERIPHERAL VASCULAR DISEASE

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PEOPLE suffering from the consequences of organic disease of the peripheral arteries such as arteriosclerosis or thromboangiitis obliterans are generally believed to be beyond the reach of surgical aid with the exception of some few in the early stages of the latter disease. Our belief is an opposing one; these people can be helped, because there is in almost every case a functional spastic factor in addition to the organic one, and this spastic factor can be removed by surgery. This report tells of operations in fifty-nine patients who have been treated by lumbar sympathectomy, the majority of whom had advanced organic peripheral vascular disease. This work was started in 1939 and represents the first attempt in a reasonably large series of cases to disprove the current fallacy of not subjecting to operation patients with advanced peripheral arteriosclerosis. Our reward has been twofold: (1) preservation of many limbs and lives through the prevention of the onset or the spread of gangrene; (2) the relief of distressing pain.

It is a well-known fact that there are certain functional disorders of the nervous mechanism of the blood vessels causing either a vasoconstriction or a vasodilatation and that the disorders causing vasoconstriction can be dramatically relieved by surgical removal of the sympathetic nerve fibers involved. The disease which probably represents the purest type of abnormal vasoconstriction is that described by Raynaud in 1862, characterized by local asphyxia and symmetrical gangrene of the extremities. Since in this disease the peripheral blood vessels are normal except for spasm, sympathectomy gives immediate relief.

What is not generally appreciated, however, is that many of the organic diseases of blood vessels have an associated vasomotor spasm which may be the cumulative factor responsible for the ensuing gangrene. The death of the part affected is due many times not to the original arterial

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disease but to the superimposed constriction of accompanying smaller vessels. Recognition of this fact and subsequent operative removal of the nervous mechanism causing the vasoconstriction will in proper cases result in the curing of pain and the actual salvation of the part from death from gangrene. It seems almost incredible that in the presence of advanced organic arterial disease the circulation of the part supplied by these vessels can be effectively aided by any operative interference. We cannot heal the first of the factors responsible for the gangrene or the threat of gangrene, the disease of the arterial coats, but we can relieve the second factor responsible, the vasomotor spasm of the neighboring smaller vessels.

A dogmatic classification of peripheral vascular disease from the clinical and pathologic point of view is routinely given in any comprehensive discussion of the subject, but it is important to bear in mind that many patients will not fall into one of the well-differentiated and generally accepted classifications but will present findings characteristic of more than one. A close study of Raynaud's original description, for instance, shows that some of his cases were organic rather than functional in origin. A useful classification is that given by Brown and Henderson.

CLINICAL CLASSIFICATION OF ARTERIAL VASCULAR DISEASE

- Functional or vasomotor types
  - Local distribution
    - Vasoconstricting types
      1. Multiple-phase color reaction: Raynaud's disease
      2. One-phase color reaction; acrocyanosis, dead finger, local syncope
    - Vasodilating types: Erythromelalgia
  - General distribution
    - Vasoconstricting types: Primary or essential hypertension, early stages
    - Vasodilating types: Primary or essential hypotension
- Organic types
  - Local distribution
    1. Arteriosclerosis, with or without thrombosis; diabetic gangrene
    2. Thromboangiitis obliterans (Buerger's disease)
    3. Simple thrombosis or embolism
    4. Arteritis of known infectious origin (rheumatic syphilitic)
    5. Aneurysm with or without thrombosis
  - General distribution: Arteriosclerosis
    1. Primary
    2. Secondary to hypertension

The functional vasoconstricting lesions of local distribution in this list are known to be improved following sympathectomy. Even certain types of functional vasoconstricting lesions of general distribution are relieved by sympathectomy. The great organic group, however, is not generally supposed to be susceptible to the sympathetic approach. Our present study concerns this latter group of organic types of peripheral arteriovascular disease; attacks on it are now being made not

frontally by attempting to alter organic changes already present in the vessels, but by a flanking movement of stopping the accompanying vasoconstriction by removing the sympathetic control. The old hopeless attitude of palliative treatment to local ischemic lesions and of passive acquiescence in frequent amputations of the toes, feet, or legs no longer is justified.

The correct differentiation of the vascular disturbances of the extremities must be made in each patient. It is of great importance to know why an ulcer is present on a toe, why the feet are cool or pale, why they sweat, why there are muscular pains in the legs, why the leg is more comfortable in a dependent position. The most important single step in the examination, the sine qua non of the differential diagnosis, is the search for pulsation in all palpable arteries. The role played by vasomotor contraction must next be determined by one or several of various tests. Since the nerve path for vasomotor contraction and for sweating is the same—over the sympathetic nerves arising from the first thoracic to the second lumbar spinal nerves—these tests are all dependent on the relative increase in blood flow and decrease in sweating of the involved extremity following release of vasomotor contraction.

Warming the rest of the body generally or warming extremities other than those to be tested by application of warm blankets or by immersion in hot water will through reflex inhibition of the tone of the vasoconstrictor center, cause dilatation of the vessels in the involved extremity and a consequent rise in temperature and computable blood flow if vasomotor spasm has been an appreciable factor in the disease of the extremity. These same effects may be produced by interrupting the vasomotor control of the extremity either by novocain injection of the sympathetic ganglia or by intrathecal injection (spinal anesthesia). A favorable local result is evidenced by a rise in temperature, a more healthy, rosy color to the skin, an absence of sweating, and a subsidence of pain. Fine determination of these results can be made by various calorimetric, oscillometric, and plethysmographic records.

If by such tests a diseased extremity shows definite circulatory improvement, a sympathectomy must be considered seriously. However, if the tests are not followed by improvement, it is generally believed that this operation would prove futile, but the latter conclusion is not justified. It has been our experience that a splendid result may follow sympathectomy in some patients whose preoperative tests gave no promising indications. Case 4 and Case 58 illustrate this point. Case 58 was of a man of 36 years, with an arterial occlusion of the vessels in the lower right leg, who showed no increase in temperature after reflex warming. Nevertheless, sympathectomy relieved all pain immediately, permitted elevation of the leg in any position without pain, and was followed by rapid healing of an ulcer of the heel of many months' duration. This man showed a symptom, common to many with this trouble, of less pain

TABLE I

ARTERIOSCLEROSIS

NO.	NAME	SEX	COLOR	AGE	COMPLAINT	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES LEGS	OPERATION	SIDE	DATE	RESULT
4	J. B.	M	W	57	Threatened gangrene left foot, intermittent claudication left leg		Left 0 Right, slight	Lumbar sympathectomy	Left	10/ 3/41	Greatly improved
5	W. B.	M	B	47	Gangrene foot, right	Arteriosclerosis generalized, gangrene toe, alcoholic neuritis	No record	Lumbar sympathectomy	Left	9/12/39	Improved
6	W. B.	M	W	67	Intermittent claudication, burning pain in foot; ulcer, toe left foot	Arteriosclerosis generalized; diabetes, mild; ulcer, foot, left	Left, slight Right 0	Lumbar sympathectomy	Left Right	3/13/40 3/28/40	Greatly improved
19	J. G.	M	W	57	Cellulitis; gangrene, right foot	Arteriosclerosis generalized; gangrene, feet, bilateral	Left 0 Right 0	Lumbar sympathectomy	Right Left	7/10/40 11/19/40	Not improved
23	B. J.	M	B	51	Pain left foot; small area gangrene, left great toe	Arteriosclerosis generalized; syphilis (Wassermann); amputation leg, right, old	Left, slight	Lumbar sympathectomy	Left	12/20/39	Greatly improved
24	K. K.	F	W	65	Severe pain, right foot; ulcer, right ankle	Arteriosclerosis generalized; ulcer, leg, right	Left 0 Right 0	Lumbar sympathectomy	Right	6/ 5/40	Greatly improved
25	J. K.	M	W	57	Intermittent claudication, severe pain right foot and ankle, ulcers right ankle	Thrombosis, arteries, leg, right	Left, slight Right 0	Lumbar sympathectomy	Right	7/26/41	Greatly improved
26	H. L.	M	W	64	Ulcers and pain both feet	Myocarditis; osteomyelitis, phalanges, feet	No record	Lumbar sympathectomy	Left Right	9/20/40 9/28/40	Greatly improved

28	C. M.	M	W	60	Intermittent claudication; gangrene, fourth toe, right foot	Arteriosclerosis generalized; gangrene, toe	Left 0 Right 0	Lumbar sympathectomy	Left Right	8/30/39 10/ 6/39	Died
31	J. M.	M	W	54	Intermittent claudication		Left 0 Right 0	Lumbar sympathectomy	Left	3/12/40	Improved
34	P. N.	M	W	57	Intermittent claudication	Arteriosclerosis generalized; varicose veins, legs, bilateral	Left, slight Right, slight	Lumbar sympathectomy	Right	10/ 7/42	Improved
35	C. N.	M	W	57	Intermittent claudication	Arteriosclerosis generalized; gangrene, foot, left; amputation, leg, right, old	Left 0	Lumbar sympathectomy	Left	6/18/40	Not improved
38	J. P.	M	W	63	Pain, right foot; gangrene, right foot	Arteriosclerosis generalized; hemiplegia, left, old; hypertension, gangrene, foot, right	Left 0 Right 0	Lumbar sympathectomy	Right	9/ 6/39	Died
40	H. R.	M	W	67	Intermittent claudication; gangrene, right foot		Left 0 Right 0	Lumbar sympathectomy	Right	12/15/39	Died
41	K. R.	M	W	67	Intermittent claudication		Left 0 Right 0	Lumbar sympathectomy	Right Left	7/ 9/40 7/19/40	Improved
42	M. S.	F	W	46	Cellulitis and gangrene	Diabetes; gangrene, foot, right	Left 0 Right 0	Lumbar sympathectomy	Right	2/ 9/40	Improved
44	C. S.	M	W	65	Pain, left leg; ulcers, left leg	Arteriosclerosis, generalized	Left 0 Right 0	Lumbar sympathectomy	Left	8/30/41	Improved
45	J. S.	M	W	62	Pain, right leg and foot; ulcer, right great toe	Arteriosclerosis generalized; diabetes, mild; gangrene, foot, right	Left 0 Right 0	Lumbar sympathectomy	Right Left	5/ 7/40 5/16/40	Not improved
48	H. S.	M	W	74	Intermittent claudication	Arteriosclerosis generalized; amputation, leg, right, old	Left 0	Lumbar sympathectomy	Left	10/14/40	Greatly improved

TABLE I—CONT'D

NO.	NAME	SEX	COLOR	AGE	COMPLAINT	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES LEGS	OPERATION	SIDE	DATE	RESULT
51	A. T.	M	W	59	Pain, right leg and foot	Arteriosclerosis generalized; hemiplegia, right; thrombosis, arteries, lower leg, right	Left, slight Right 0	Lumbar sympathectomy	Right	5/21/40	Improved
53	G. T.	M	W	60	Gangrene, left foot	Arteriosclerosis generalized; diabetes; gangrene, foot, left	Left 0 Right 0	Lumbar sympathectomy	Left	3/27/40	Improved
55	A. W.	F	W	37	Coldness, foot, left	Diabetes; amputation, leg, right, old; gangrene, foot, left	Left 0	Lumbar sympathectomy	Left	12/ 6/39	Greatly improved
57	R. W.	M	W	57	Pain in right foot and leg	Arteriosclerosis generalized; myocarditis; thrombosis, arteries, leg, lower, right	Left, slight Right 0	Lumbar sympathectomy	Right	10/22/40	Improved
17	W. G.	M	B	59	Coldness and pain in left foot	Ulcer, foot, left	Left 0 Right 0	Lumbar sympathectomy	Left	7/13/40	Died

In the group classified as Arteriosclerosis there were 24 patients, 20 males and 4 females, a ratio of 5 to 1. Two of the 4 females had diabetes and gangrene of the feet. It is interesting to observe how much more frequent is the incidence of peripheral vascular trouble in the male and that in the 4 female patients, gangrene was present only in the 2 with diabetes. There were 3 Negro patients in the group of 24, all male. The infrequent occurrence of this disease in Negroes, 1 to 8 ratio, in a hospital where the ward cases are almost equally divided between the two races, and the absence of a single case in Negro women are two additional interesting observations. The average age of these 24 patients was 58.3 years. Of the 10 patients with intermittent claudication, 5 were entirely or largely relieved of this symptom. Of the 18 patients with ulceration or gangrene, in 11 healing took place following sympathectomy, 3 had subsequent amputations, and 4 died. In the total of 24 patients, 16 showed preoperatively no pedal pulse, 6 showed a faint unilateral pulse, 1 a faint bilateral pulse, and in 1 no record of pulsation was recorded. Following operation in 1 patient a pedal pulse appeared which was not present before operation. In 3 patients an amputation was necessary in spite of a preceding sympathectomy. Four patients had had a previous amputation of one leg before coming to us. By a prophylactic lumbar sympathectomy we were able to save the remaining leg in 3 of these 4 people. The final result showed 8 patients greatly improved, 9 improved, 3 not improved, and 4 died. These were the only deaths in our series, all four occurring in the arteriosclerosis group. The review of the histories in the cases with death, as given in detail earlier in this communication, shows that we may have been a little too ambitious at the start, but we were dealing with desperate situations. This particular group of 24 patients is the type which has heretofore almost always been considered hopeless.

with the foot in the dependent position and more pain according to the degree of elevation; he could sleep only sitting up with the foot hanging down over the side of the bed. It seems strange that the dependent foot, becoming dusky red and engorged, should grow less painful. Evidently in all positions of the extremity the arterial supply is equally poor; in the elevated position the extremity is also deprived of a large amount of its venous blood, but in the dependent position there is of course some venous stasis and even this relatively stagnant venous blood carried sufficient oxygen to be of some assistance to oxidation in the tissues and to consequent relief of pain.

The selection of patients for this operation requires the greatest care in balancing the possible benefits against the risk incurred when people in this age and morbidity group are subjected to any operation. Manifestly it would be wrong to employ this operation indiscriminately in patients suffering from diminished arterial supply to the extremities who show no evidence of improvement after preliminary tests for vasomotor spasm. Nevertheless, many patients in this group will be benefited by sympathectomy and, if their physical condition permits, should be subjected to the operation. Likewise, manifestly it is right to employ this operation in all patients suffering from the serious consequences of diminished arterial supply to the extremities, whose tests show that vasomotor spasm as well as actual organic arterial disease is a causative factor, provided the physical condition of the patient permits the operative risk.

Fortunately the operative risk is not, or should not be, great. Our rule is that in no case is a bilateral resection done at the same operation, that for the lumbar resection spinal anesthesia is by all odds the best anesthesia, and that patients whose physical condition prohibits the use of spinal anesthesia should not be subjected to this operation under any type of anesthesia.

In fifty-nine patients there were eighty-two lumbar sympathectomies (eighteen of the right side, twenty-two of the left side, and twenty-one bilateral). There were four operative deaths. The first death (Case 17) was of a Negro aged 59 years, a patient thought to have pulmonary tuberculosis, who developed a cold, painful, pulseless left foot with an ulcer on the dorsum. Lumbar sympathectomy did not benefit his foot, and he died four weeks later, autopsy showing squamous cell carcinoma of the bronchus of the left lung, gangrene of the left leg, and bronchopneumonia. The second death (Case 28) was of a white man aged 60 years, with generalized arteriosclerosis, a four-year history of intermittent claudication, and beginning gangrene of the fourth toe of the right foot. Bilateral sympathectomy, done one week apart, resulted in great improvement, but he died suddenly two weeks after the last operation with coronary thrombosis. The third death (Case 38) was of a white man 63 years of age, with generalized arteriosclerosis, a partial

TABLE II

THROMBOANGIITIS OBLITERANS

NO.	NAME	SEX	COLOR	AGE	COMPLAINT	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
1	T. A.	M	W	34	Intermittent claudication; rest pain, right foot; phlebitis, right leg	Amputation, leg, left, old; phlebitis, leg, right	Right 0	Lumbar sympathectomy	Right	6/ 4/40	Greatly improved
2	M. A.	M	W	52	Intermittent claudication		Left 0 Right 0	Lumbar sympathectomy	Right Left	1/ 2/41 2/17/41	Improved
3	C. B.	M	W	50	Intermittent claudication	Hypertensive vascular disease; bronchiectasis, post-infectional	Left, slight Right, slight	Lumbar sympathectomy	Right	12/12/40	Improved
8	W. B.	M	W	24	Intermittent claudication	Varicose veins	Left + Right +	Lumbar sympathectomy	Right Left	8/21/41 8/28/41	Greatly improved
10	G. C.	M	W	36	Cellulitis and phlebitis	Cellulitis and phlebitis, legs, bilateral	Left 0 Right 0	Lumbar sympathectomy	Right Left	7/ 7/40 7/16/40	Greatly improved
11	N. C	M	B	31	Intermittent claudication	Varicose veins, leg, left	Left 0 Right 0	Lumbar sympathectomy	Left	10/ 2/41	Greatly improved
14	E. D.	F	W	37	Intermittent claudication; ulcer, foot, left; rest pain, foot, left		Not recorded	Lumbar sympathectomy	Left	6/ 9/41	Greatly improved
15	A. F.	M	B	39	Intermittent claudication; gangrene, foot, left	Hypertensive vascular disease; gangrene, foot, left	Left 0 Right 0	Lumbar sympathectomy	Left	2/12/41	Greatly improved

16	A. F.	M	W	40	Pain, right foot		Left 0 Right 0	Lumbar sympathectomy	Right	9/14/40	Improved
20	A. H.	M	B	40	Intermittent claudication		Left 0 Right, slight	Lumbar sympathectomy	Left Right	10/22/41 10/27/41	Improved
21	L. H.	M	W	48	Intermittent claudication		Left 0 Right 0	Lumbar sympathectomy	Right	8/26/41	Improved
27	J. L.	M	W	48	Intermittent claudication		Left 0 Right 0	Lumbar sympathectomy	Left Right	10/16/40 10/29/40	Improved
37	L. P.	M	B	30?	Pain and swelling in left leg and foot	Gangrene and osteomyelitis, toes, left foot	Right, slight Left 0	Lumbar sympathectomy	Left	3/15/40	Not improved
43	D. S.	M	W	49	Intermittent claudication		Left 0 Right 0	Lumbar sympathectomy	Right Left	1/23/41 11/19/41	Improved
54	I. V.	M	W	38	Intermittent claudication; pain in hands; rest pain in left foot		Left 0 Right +	Lumbar sympathectomy	Left	8/ 9/41	Improved

In the group classified as Thromboangiitis Obliterans there were 15 patients, 14 males and 1 female. Four of the 14 males were Negroes, the 1 female was white. The average age in this group was 40.2 years. Of the 12 patients with intermittent claudication, 9 were either completely or largely relieved of the complaint. Of the 4 patients with ulceration or gangrene, in 3 healing occurred following sympathectomy; the fourth required amputation in spite of sympathectomy. In the total of 15 patients, 9 showed preoperatively no pedal pulse, 3 a faint bilateral pulse, and in 1 no record of pulsation was recorded. Following operation a pedal pulse appeared in 5 patients, which was not obtainable before operation. One patient had had a previous amputation before coming to us (Case 1). His remaining leg was gravely threatened but responded wonderfully to operation, even showing a strong pedal pulse not present before. The final result showed 6 patients greatly improved, 8 improved, 1 not improved, no deaths.

hemiplegia of six years' standing, hypertension, and pain with gangrene in the right foot. He requested operation because he had seen such improvement on two other ward patients, in spite of our advice to the contrary. Against our better judgment we consented. Because of his hypertension, 210/108, the operation was performed under ether inhalation instead of our routine spinal procedure. He developed atelectasis of the lower lobe of the right lung, which soon involved the entire lung, and he died with bronchopneumonia four days after operation. The fourth death (Case 40) was of a white man aged 63 years, with generalized arteriosclerosis, red, cold hands with loss of sensation in the finger tips, and cold pulseless feet with early gangrene of the first, third, and fourth toes of the right foot with inflammation spreading up the foot. Knowing that an amputation was imminent unless the gangrenous process could be stopped, a lumbar sympathectomy was done, Dec. 15, 1939, without avail. The gangrene continued to develop, a mid-thigh operation was done three and one-half weeks after the sympathectomy. He died several days following amputation, autopsy showing almost entire occlusion of the left anterior coronary artery, and extensive myocardial necrosis.

The operation of sympathectomy attacks the disease in several ways. Extremities with poor arterial circulation are peculiarly susceptible to cold; the colder they become the more constriction takes place in the vessels, and, since the nervous pathway for vasoconstriction and sweating is the same, the more the vessels become constricted the more sweating takes place with further lowering of temperature. This vicious cycle is broken by operation in two places; the vasoconstrictor fibers are paralyzed and sweating is abolished. In several patients an increase in the temperature of the leg and an improvement in its color were found on the side opposite to, as well as the side of, operation. This is probably due to some crossed innervation of the sympathetic fibers.

Gangrene and pain, alone or in combination, are the challenging factors in this problem. In our total of fifty-nine patients, thirty-seven (62.7 per cent) had ulceration or gangrene, while forty-six (77.8 per cent) complained of pain.

GANGRENE

The rapid healing of a previously indolent ulcerated or gangrenous area will often follow sympathectomy. The result in Case 4 was most gratifying, representing a type believed hopeless under the generally accepted standards of operability. The patient was a man 57 years of age, with tingling and burning pain in the pulseless left foot and cyanosis of the toes. Preliminary tests by novocain injection of the sympathetic trunk were discouraging. Following sympathectomy, the purplish toes were restored to good color, pain ceased, and the foot became warmer. Healing occurred (Case 23) in a Negro, aged 51 years,

TABLE III

FROSTBITE GANGRENE

NO.	NAME	SEX	COLOR	AGE	COMPLAINT	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
7	J. B.	M	W	46	Frostbitten feet	Arteriosclerotic heart disease; gangrene, frostbite, feet, bilateral	Left + Right +	Lumbar sympathectomy	Left	5/ 7/40	Improved
13	O. C.	M	B	46	Frostbitten feet	Gangrene, frostbite, feet, bilateral; syphilis (Wassermann)	Left, slight Right, slight	Lumbar sympathectomy	Left	3/14/40	Improved
29	O. M.	M	W	57	Frostbitten feet	Myocarditis; auricular fibrillation; gangrene, frostbite, foot, left	Not recorded	Lumbar sympathectomy	Left Right	5/23/39 5/23/39	Improved
32	F. M.	M	W	67	Frostbitten feet	Arteriosclerosis, generalized; gangrene, frostbite, foot, right	Left, slight Right, slight	Lumbar sympathectomy	Right	5/18/40	Improved
36	J. P.	M	W	64	Frostbitten feet	Arteriosclerosis, generalized; gangrene, frostbite, foot, left, old; amputation, leg, right, traumatic, old	Left 0	Lumbar sympathectomy	Left	4/24/40	Not improved
46	J. S.	M	W	67	Frostbitten feet	Arteriosclerosis, generalized; gangrene, frostbite, foot, right; amputation, leg, left, old	Right 0	Lumbar sympathectomy	Right	2/23/40	Improved

In the group classified as Frostbite Gangrene there were 6 patients, all male, 5 white and 1 Negro, the average age being 57.8 years. All 6 had ulceration or gangrene, of which 4 healed after sympathectomy. Two patients had had a previous amputation of one leg; the remaining leg in 1 was saved by sympathectomy. There were two legs amputated after sympathectomy but in both these cases the sympathectomy was done to limit the extent of the gangrene and not in the expectation to save the leg. The final result showed 5 patients improved, 1 not improved, and no deaths.

TABLE IV

VASOSPASM AND OBLITERATIVE ARTERITIS FOLLOWING EXPOSURE TO COLD

NO.	NAME	SEX	COLOR	AGE	SYMPTOMS	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
39	B. R.	M	B	45	Pain in both feet	Syphilis (Wassermann)	Left 0 Right +	Lumbar sympathectomy	Left	2/17/40	Improved
49	J. S.	M	W	47	Pain in both feet	Amputation, toes, feet, bilateral, old; ulcer, heel, left	Left + Right +	Lumbar sympathectomy	Left Right	10/21/39 12/ 6/39	Improved
59	W. W.	M	W	44	Pain in both feet	Amputation, toes, feet, bilateral, old	Left 0 Right 0	Lumbar sympathectomy	Right	3/ 5/40	Improved
30	W. M.	M	W	59	Frostbitten feet	Hypertension; cardiovascular disease; ulcer, great toe, foot, left	Left, slight Right +	Lumbar sympathectomy	Left Right	1/ 2/39 3/29/39	Improved

The classification Vasospasm and Obliterative Arteritis Following Exposure to Cold, or Cold-sensitive Extremities, includes a group of patients who, after an exposure to a cold temperature sufficient to cause mild or severe frostbite, are rendered by that exposure always very susceptible to subsequent exposure to cold temperature. These patients do not have generalized peripheral vascular disease. In civil life many of these patients suffer with chronic alcoholism and have wandered about the snow and slush of streets for many hours or days between visits to taverns. In the army a great many of these people will be found as a result of the rigors of winter fighting. The exact pathologic state of the arteries has not been described. Usually there is good pulsation in the feet, and always great spasm of the arteries can be demonstrated by test.

Four of our patients fall into this classification, 3 white males, and 1 Negro male, the average age being 48.7 years. All 4 had warm, painless extremities after operation. In none of them was there an absence of pedal pulsation even before operation, and in none was an amputation necessary.

who had suffered a leg amputation because of arteriosclerotic gangrene two years previously and whose remaining leg in which gangrene had started was certainly saved by sympathectomy. Healing also occurred (Case 25) in a man 57 years of age, in whom painful ulcers of long standing healed promptly. In Case 42, there was diabetes, arteriosclerosis, advancing gangrene of the right foot, and fever in a woman aged 46 years; following sympathectomy there was a rapid subsidence of fever and rapid healing of the foot. In Case 58, a 36-year-old man with a pulseless right foot, had for two years pain in the toes, arch, and heel of the foot, and for four months a painful ulcer of the heel which had resisted all efforts at cure. Sympathectomy brought immediate relief of all pain and rapid healing.

One leg had been amputated previously in four of our cases (Cases 1, 23, 48, and 55). By sympathectomy on the opposite side we believe that we saved the remaining leg from a similar fate. Even the presence of old age and advanced arteriosclerosis did not deter us, as in one man of 74 years (Case 48), where remarkable improvement of the remaining leg followed operation. In Cases 19, 35, 40, and 45 sympathectomy failed to save the leg.

With advanced frostbite gangrene, sympathectomy failed to save the leg, but probably limited the level of the gangrene in Cases 13 and 36. A less advanced type of frostbite gangrene was helped by sympathectomy in Cases 29, 49, and 59.

PAIN

The relief of various types of pain following lumbar sympathectomy has been gratifying and most instructive. In such a group of patients are found (1) the pain of intermittent claudication in the calf muscles of the leg, (2) the diffuse rest pain in a foot with or without the presence of actual ulceration, and (3) the rest pain localized in a small ulcer or in a gangrenous toe.

Twenty-four of our patients suffered with intermittent cramplike pain in the leg muscles after exercise. This type of pain is not generally supposed to be amenable to any form of therapy. Nevertheless, following operation nine patients were relieved of all pain, eight were improved, and seven were unimproved. In three of the seven patients unimproved for intermittent claudication in the calf muscles, the circulation of the foot was improved as evidenced by the rise in temperature, the improvement in color, and the cessation of pain in the foot, as in Cases 27, 34, and 43.

Dr. R. W. Wilkins and we decided that Case 41 would be a particularly favorable one from which valuable information could be obtained in this unanswered question of the possibility of operative relief in intermittent claudication. The patient was an intelligent woman 64 years of age, the wife of a retired army colonel. Her feet were cold and pulse-

TABLE V

VARICOSE ULCER

NO.	NAME	SEX	COLOR	AGE	SYMPTOMS	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
18	J. G.	M	W	58	Ulcer, leg, left		Right + Left +	Lumbar sympathectomy	Left	3/ 5/40	Not improved
22	J. I.	M	W	64	Ulcer, leg, left	Arteriosclerosis; heart disease	Left + Right +	Lumbar sympathectomy	Left	1/10/41	Not improved
50	J. S.	M	W	64	Ulcer, both legs	Arteriosclerosis, generalized	Not recorded	Lumbar sympathectomy	Right Left	4/15/40 4/25/40	Improved
56	D. W.	F	B	40	Ulcers, both legs		Left + Right +	Lumbar sympathectomy	Left Right	7/17/40 7/27/40	Not improved

Four patients with varicose ulcers were subjected to lumbar sympathectomy because it was believed that a warmer, dryer skin would tend to heal more rapidly. In 3 of these patients, following ligation and injection of the varicose veins, a lumbar sympathectomy was done and rapid healing of the ulcers resulted. However, the ulcers recurred, so they were excised together with their surrounding avascular fibrous edges and on their base were placed small deep grafts. Permanent healing took place in all 3. The fourth patient (Case 50) had ulcers on both legs which healed quickly and remained healed following sympathectomy, but he died of pulmonary tuberculosis several months after operation. Our impression in this group of persistent varicose ulcers is that the method of procedure should be first the treatment of the varicose veins, with excision and grafting of the ulcer, and then, if judged advisable, a lumbar sympathectomy, which undoubtedly would tend to prevent recurrence of the ulcer.

TABLE VI

OBSTRUCTION VENA CAVA

NO.	NAME	SEX	COLOR	AGE	SYMPTOMS	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
12	A. C.	M	W	24	Swelling of legs; ulcers, both legs		Left + Right +	Lumbar sympathectomy	Left Right	7/31/40 8/14/40	Improved

One patient (Case 12) had, for six years, edema of both legs and signs of lower vena caval obstruction following what was apparently a phlebitis of the pelvic veins. Exploratory laparotomy showed obliteration of the lower portion of the vena cava, inferior. A bilateral lumbar sympathectomy gave him much relief, due no doubt to the relief of circulatory spasm present in patients who have suffered with phlebitis.

less, and she had for five years suffered severe cramplike pains in the calves of her legs after walking a short distance. Following bilateral lumbar sympathectomy both feet became pink and warm. Her description of the subjective results is quoted because it is significant.

March 13, 1941, eight months after operation, she wrote, "I have no severe cramps. My legs and feet have been much warmer. I cannot walk more than two short blocks before I find the blood pumping and find it difficult to go on. The bottoms of my feet burn and smart when I stand too long." Sept. 17, 1941, fourteen months after operation, she wrote, "I have *no* cramps in my legs. The flesh is sensitive and my legs and feet ache at times but I am certainly thankful not to have cramps." Jan. 24, 1943, two years 6 months after operation, she wrote, "I cannot walk more but have no cramps, for which I am most grateful to you."

In the fall of 1939, Wilkins first made the observation to the senior author that by sympathectomy it seemed we were often substituting for the pain of claudication in the legs a sensation of feeling "tired" in these muscles. In clinics before the staff and students this author has many times since referred to this observation. Cases 2 and 6 are also illustrative of this change. In Case 6, a man aged 67 years was relieved of all pain of intermittent claudication, to have it replaced by a tired feeling.

Sometimes, although the foot is warmer and dryer and pinker after operation, and free from local pain, there is no improvement in the intermittent claudication. However, even in these patients, a warmer, dry, pink foot results, giving evidence of better circulation in the skin, making the foot less liable to become excoriated or fissured, and, therefore, less susceptible to fungus or other infection which may so often be of tragic consequence.

The second type of pain, the relief of which has proved most encouraging, is the diffuse rest pain in a foot with or without the presence of actual ulceration. Such pain was seen in twenty-nine of our patients. By sympathectomy this pain was completely relieved in sixteen patients, and partially relieved in seven patients. It should be especially remarked that ten of the patients in the group of those with diffuse rest pain in an extremity also suffered with intermittent claudication. The dramatic, immediate release from pain in these patients is almost as gratifying to the operator as to the patient. Formerly we felt helpless with the problem of the patient who sat a great part of the day and night on the edge of his bed, rocking back and forth, rubbing his foot in an effort to stop the pain. All medical and physiotherapeutic aid proved unavailing. We now feel certain that we can promise the great majority of these patients immediate relief by sympathectomy. Cases 14, 24, 25, 54, and 57 are striking examples. A woman 65 years of age, with a two-year history (Case 24) of relentless pain in the right foot and ankle,

TABLE VII

IDIOPATHIC THROMBOSIS, ARTERIES, LEG

NO.	NAME	SEX	COLOR	AGE	SYMPTOMS	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
9	T. B.	M	W	48	Intermittent claudication	Thrombosis, arteries, leg, left	Left 0 Right +	Lumbar sympathectomy	Left	7/16/41	Improved
58	E. W.	M	W	36	Rest pain, right foot; ulcer, right heel	Thrombosis, arteries, leg, right	Left + Right 0	Lumbar sympathectomy	Right	4/26/40	Greatly improved

A separate classification was called Idiopathic Thrombosis, Arteries, Leg, to include those patients who showed no signs of generalized vascular disease, and who had one leg in vascular difficulty for some unexplained reason with the other leg normal. The first patient, a man 48 years of age, showed, after operation, improvement in the color and temperature of the leg but only slight relief to intermittent claudication. The second patient obtained immediate and dramatic relief of pain in his leg and foot with rapid healing of a long-standing ulcer.

had cold pulseless feet. A man aged 57 years (Case 25) suffered severe rest pain in his right foot which made sleep impossible unless the leg hung down outside his bed. A man aged 57 years, with angina pectoris (Case 57), had been suffering with sudden severe pain in his right leg and foot over a period of two months, and even with hypodermic injections of morphia he could obtain only two hours' sleep at a time. Operation stopped all pain immediately, and he resumed an active law practice with only a modicum of his former pain.

The third type of pain encountered, that of rest pain localized in a small ulcer or in a gangrenous toe, is a circumscribed, intense pain which may or may not be associated with the pain of intermittent claudication or that of diffuse pain in the affected extremity. Four of our cases are particularly good examples of this local pain, Cases 14, 24, 25, and 58. All of these patients showed immediate relief by sympathectomy. In two women (Cases 14 and 24), aged 37 and 65 years, each with a small agonizingly painful ulcer of the leg, the pain ceased immediately and the ulcer healed rapidly following operation. A man of 36 (Case 58) showed much the same type of ulcer with the same gratifying result.

The current fallacy of not operating on patients for the relief of symptoms due to advanced arteriosclerosis has in our experience been disproved. Under the present accepted standards the majority of the patients in our group would have been considered not amenable to operative relief. However, we did operate, and a reading of the abstracts of the clinical records alone will illustrate how gratifying the results have been.

We do not wish to minimize the great importance of measures other than operative with these patients, or to maintain that such measures are not at times in themselves sufficient. The majority of our patients reached us after repeated trials with various drugs to stimulate vasodilatation, and with courses of intermittent positive and negative pressure to an extremity. Such additional aids should be continued after operation, if complete relief is not given following sympathectomy. Furthermore, an extremity, after the vasoconstrictor influence has been removed, is more susceptible than before to such treatment as that of passive vascular exercise. Of special importance with these people is the meticulous daily care given by the patient to his feet, his avoidance of injury when cutting the nails, and his avoidance of vasoconstrictor influences such as cold, mental anxiety, and tobacco.

We have included in this report patients with various complaints for which a lumbar sympathectomy was done. The clinical subdivisions have been further analyzed under the following classifications: (1) arteriosclerosis (24 cases), (2) thromboangiitis obliterans (15 cases), (3) frostbite gangrene (6 cases), (4) vasospastic extremities following old exposure to cold (4 cases), (5) varicose ulcer (4 cases), (6) obstruction

TABLE VIII

TROPHIC ULCER

NO.	NAME	SEX	COLOR	AGE	SYMPTOMS	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
33	F. M.	F	W	29	Ulcer, both feet	Alcoholic neuritis	Left + Right +	Lumbar sympathectomy	Left	3/ 1/40	Not improved

One patient with a trophic ulcer of the foot due to alcoholic neuritis and one with a chemical burn did not seem to be helped by lumbar sympathectomy.

TABLE IX

CHEMICAL BURN

NO.	NAME	SEX	COLOR	AGE	SYMPTOMS	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
52	J. T.	M	B	41	Acid burn, right foot	Ulcer, foot, right, infected; syphilis (Wassermann)	Not recorded	Lumbar sympathectomy	Right	5/28/40	Not improved

TABLE X

UNUNITED FRACTURE

NO.	NAME	SEX	COLOR	AGE	SYMPTOMS	ADDITIONAL DIAGNOSES	PULSATION IN PEDAL ARTERIES	OPERATION	SIDE	DATE	RESULT
47	R. S.	M	W	34	Broken right leg	Fracture, tibia and fibula, right, ununited	Left, slight Right, slight	Lumbar sympathectomy	Right Left	10/13/41 10/20/41	Improved

One patient with an ununited fracture of the right tibia and fibula, who had cool feet and poor pedal pulsations, showed rapid healing of the bones after sympathectomy. This observation is made more interesting by a recent patient, not reported in this series, who had the atrophy described by Sudek in one ankle and foot, and showed immediate subsidence of pain after sympathectomy with a rapid improvement in the structure of the bone.

vena cava (1 case); (7) idiopathic thrombosis, arteries, leg (2 cases), (8) trophic ulcer (1 case), (9) chemical burn (1 case), (10) ununited fracture (1 case).

As stated previously, the exact pathologic classification of certain cases is clinically impossible. What is the dividing line between thromboangiitis obliterans and arteriosclerosis? Is it the age of the patient? Is it the absence of generalized sclerosis or of x-ray changes in the arteries? Should patients with diabetes and evidence of peripheral vascular damage be given a special classification?

We have included in our group of thromboangiitis obliterans those patients 50 years of age or less who complain of intermittent claudication or of rest pain, or who have had recurrent attacks of phlebitis and who show little or no pulse in the arteries of the extremities and no evidence of a generalized sclerosis of the arteries throughout the body.

The five patients with diabetes are included in our group of arteriosclerosis since their diabetes seems to represent a causative factor superimposed on their arteriosclerosis rather than a discrete entity.

The first of the operations in this series was done Jan. 2, 1939, the last on Oct. 27, 1941, a period of two years and nine months. We had planned to allow a greater postoperative interval to elapse before making this report but, since two of us (I. R. T. and W. S. C.) are on active service in foreign countries,* it seems wisest to submit our present records for publication with the full realization that some of our patients reported as improved will subsequently have further peripheral vascular trouble. However, these patients have been given substantial relief even if for a limited period of time. Although some of the patients with thromboangiitis obliterans may have further trouble, we believe our unusually hopeful figures in this group have been due to early operation. We feel certain that we have relieved pain and saved many legs in this, and in the older, arteriosclerotic group usually regarded as hopeless, and have added a period of more comfortable living to a group of patients who have heretofore been destined to spend their remaining days in bed, in a wheel chair, and in more or less constant pain.

THE TECHNIQUE OF THE EXTRAPERITONEAL APPROACH FOR LUMBAR SYMPATHECTOMY

1. Anesthesia.

2. An incision is made through the skin of the anterior abdominal wall parallel to the midline and 5 cm. lateral to it beginning at a level 5 cm. below the umbilicus and extending directly upward a distance of 8 cm. The upper end of this incision is then continued at an obtuse angle laterally for a distance of 4 cm. (We have tried several other incisions but this one affords better exposure and requires much less muscle retraction.)

*The third author (W. R. M.) is entering the service at this writing.

3. The fascia and the lower fibers of the external oblique muscles are incised and retracted along their line of direction (Fig. 1).

4. The internal oblique and transversalis muscles are separated in the same way (Fig. 2). (The first assistant is more helpful standing on

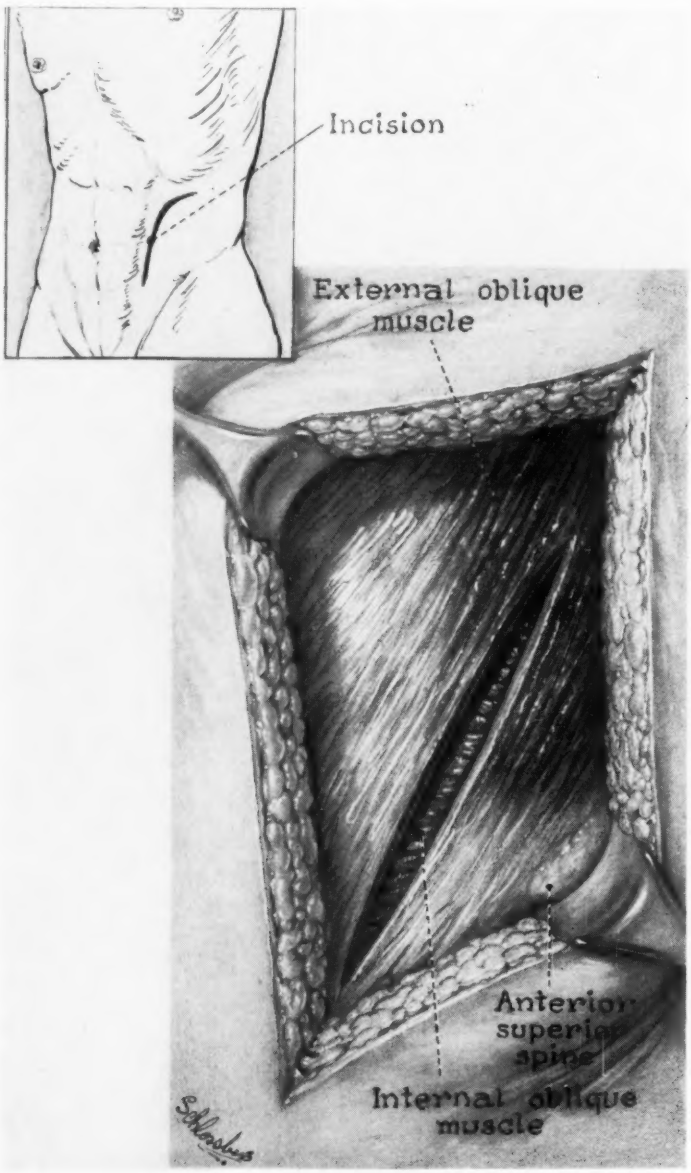

Fig. 1.

the same side of the patient as the operator, the second assistant with the retractors on the opposite side.)

5. This last step exposes the peritoneum. It is important to begin the separation between the peritoneum and the transversalis muscle laterally and not medially, and not to continue the separation too far

medially. The peritoneum is much thinner and much more closely attached to the transversalis muscle medially, and any attempt to separate them here may result in tearing through the peritoneum into the abdominal cavity (Fig. 3).

6. Separation of the peritoneum is now continued around posteriorly to the midline, great care being taken to free the peritoneum from the fat and areolar tissue over the quadratus lumborum and iliopsoas muscles.

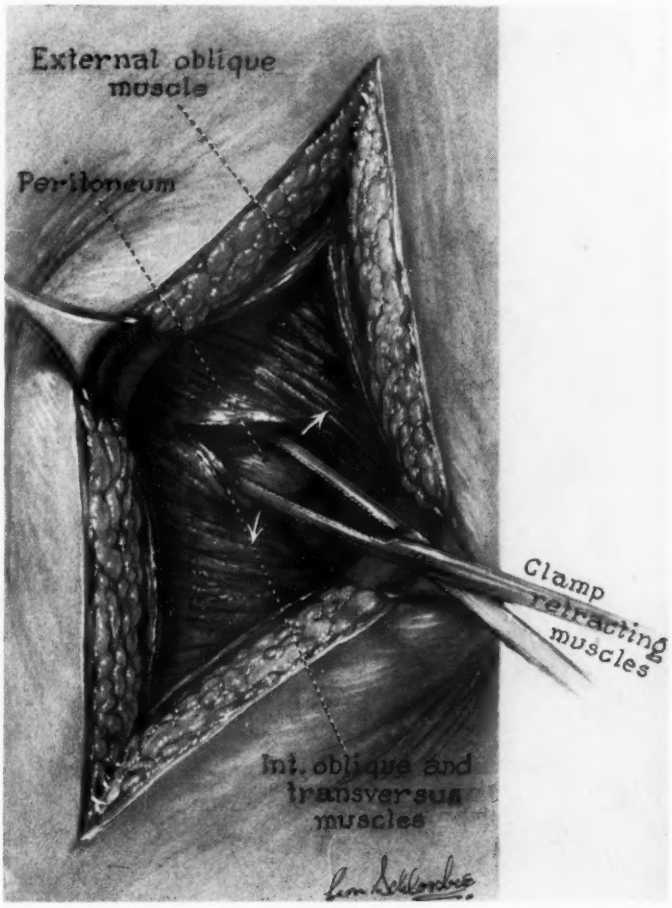

Fig. 2.

7. The sympathetic chain is next located running in its constant position against the bodies of the lumbar vertebrae and parallel to the medial border of the corresponding psoas major muscle. Location of this chain may be facilitated by palpation with the gloved finger.

8. The chain is gently retracted from its bed with a blunt hook and severed 1 cm. distal to where it crosses the brim of the true pelvis.

9. With the proximal end of the severed chain clamped and gently lifted the segment of the chain containing the second, third, fourth, and fifth lumbar ganglia is resected (Fig. 4).

10. After all bleeding points are scrupulously controlled the wound is closed in layers with fine silk.

FINDINGS

1. In patients suffering from peripheral arterial disease it is possible by lumbar sympathectomy to heal indolent ulcers quickly and to prevent the spread of gangrene and the necessity of amputation.

2. In a number of patients who have already had one leg amputated we have by sympathectomy been able to save the remaining leg—bringing about the difference between moderate disability and an almost helpless cripple.

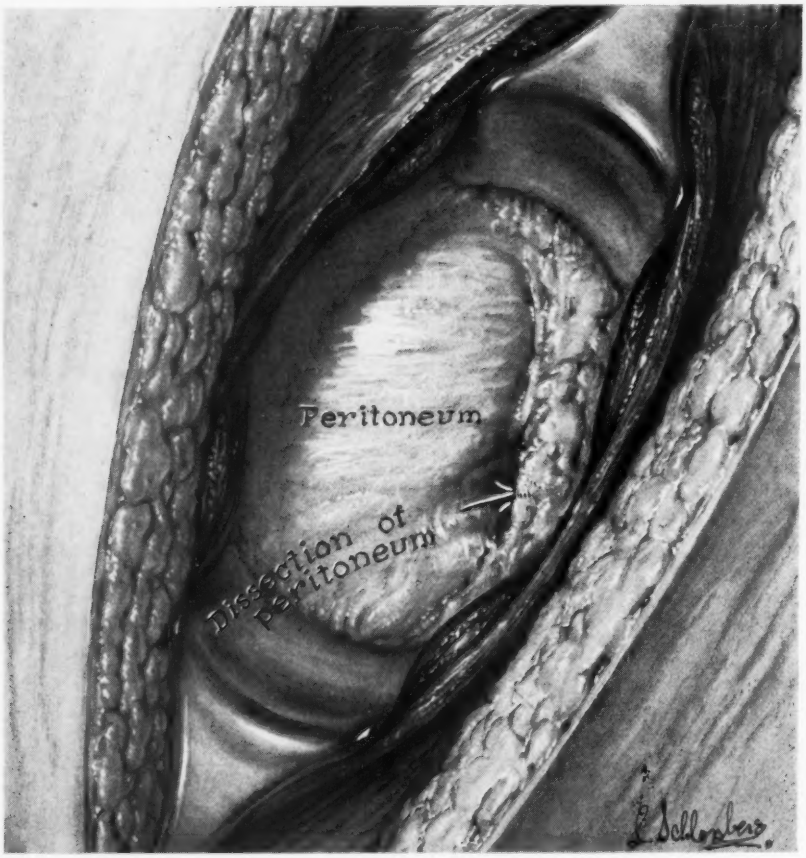

Fig. 3.

3. Patients threatened with serious vascular trouble in the extremities should have a prophylactic sympathectomy before a stage of ulceration develops.

4. Following operation the pain about an ulcer, pain in the toes, or pain in the feet disappears in the great majority of cases.

5. The muscular pain of intermittent claudication disappears in many patients following operation. Sometimes the patient will state that his

legs tire after walking a number of yards but that he has none of the old cramplike pain. In some patients the pain of intermittent claudication is not helped by operation, but the foot and leg on the side operated upon are made warmer and of a better color, and the danger of gangrene averted.

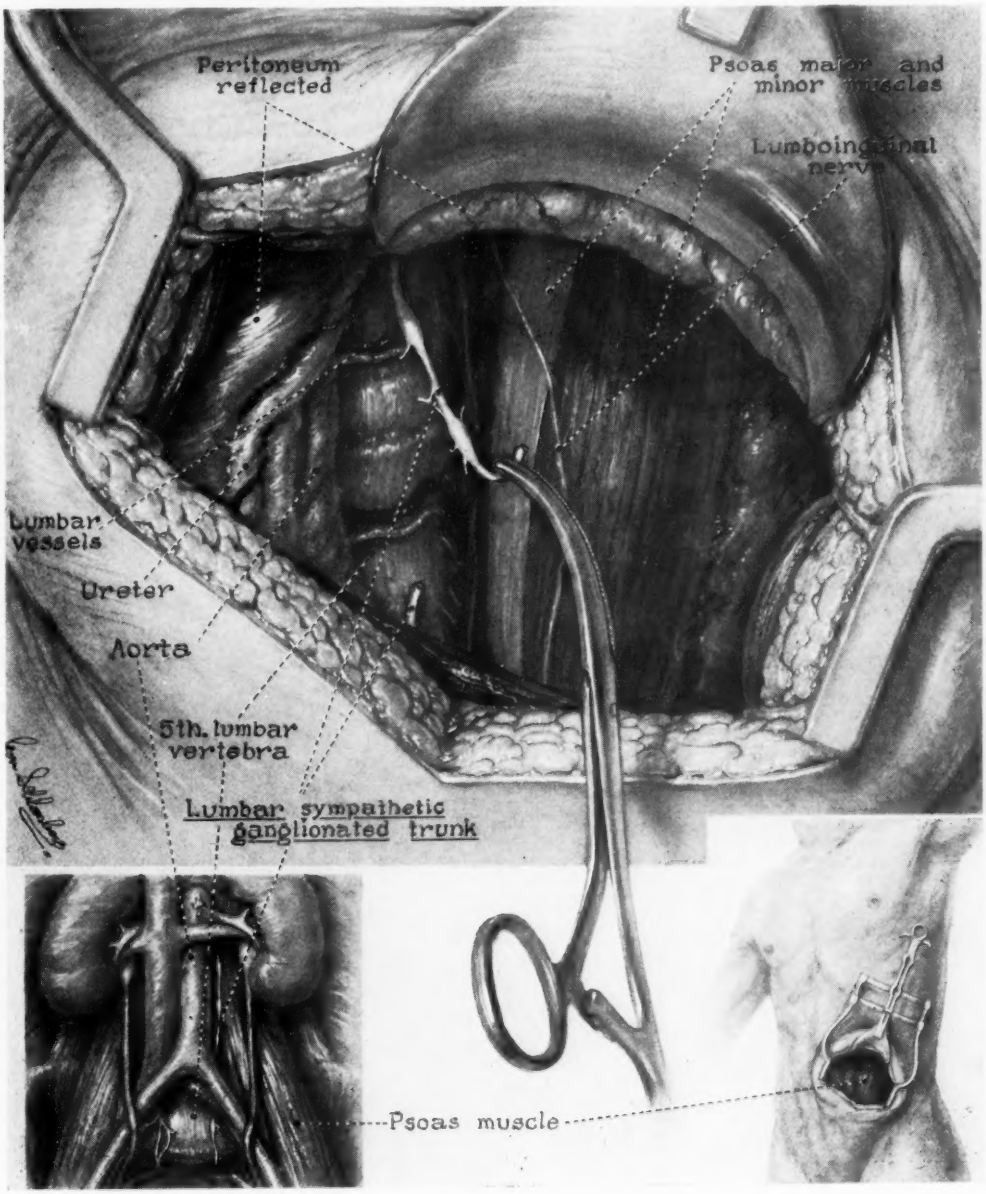

Fig. 4.

6. A negative response of the patient to preliminary tests should not preclude the possibility of aid through sympathectomy. In many patients, during a preliminary test by novocain sympathetic injection,

the temperature of the foot is not increased nor is the color of the affected toes improved, although the pain usually ceases. Nevertheless, following sympathectomy, there is a rise in temperature, a great improvement in color, and a cessation of pain.

7. The healing of varicose ulcers is not helped by sympathectomy until the ulcer with its surrounding avascular fibrous tissue is excised, the area grafted, and the varicose veins injected. The better circulation in the skin resulting from sympathectomy then helps prevent the recurrence of the ulcer.

8. Lumbar sympathectomy did not hasten the healing of an ulcer resulting from a chemical burn nor that of an ulcer due to alcoholic peripheral neuritis.

9. In one patient with delayed union of a fracture of the tibia and fibula new bone was formed at the site of injury six weeks after sympathectomy.

CONCLUSIONS

1. The association of vasomotor spasm with organic disease of blood vessels is stressed.

2. Gangrene and the threat of gangrene associated with peripheral organic disease are amenable to sympathectomy.

3. Pain in organic as well as in functional vascular disease may be relieved by sympathectomy.

4. The current fallacy of not operating on patients with advanced arteriosclerosis has been disproved.

We are especially indebted to Dr. Thomas B. Aycock, Chief of the Surgical Service of the Baltimore City Hospitals, who asked the senior author in 1939 to take charge of the patients with peripheral vascular diseases. Dr. (now Lieutenant Colonel) George H. Yeager, whom we have consulted frequently, operated on the first patient in our series. Dr. W. S. Chency first suggested that we try lumbar sympathectomy in a large group of patients with advanced arteriosclerosis. Dr. (now Lieutenant Colonel) J. Bordley III, Dr. R. W. Wilkins, and Dr. L. W. Eichna helped us a great deal in the preliminary clinical studies of our patients. Forty-five of these patients were from the public wards, fourteen from the private. The two senior authors performed the majority of the operations; the other operators, in order of number of operations done, were Drs. R. D. Woolsey, T. A. Nestor, L. K. Woodward, S. R. Gehlert, Jr., H. L. Rigdon, W. R. Moses, E. R. Haley, R. P. Bell, Jr., G. S. Bourne, H. S. Smith, Jr., H. W. Jones, and A. B. Crampton. To them we are very grateful, as well as to Drs. D. B. Hebb, B. H. Hand, and H. H. Trout, Jr., who helped with cases not included in this report during the early months of 1942. All of these men are now in the armed forces.

Book Reviews

Backache and Sciatic Neuritis. By Philip Lewin. Philadelphia, 1943, Lea & Febiger.

The author has produced a compendium of information available in the American and English literature on the subject of backache and sciatic neuritis. For this he deserves much credit. It is a big task and one which has not before been undertaken by many. There is a great need for such a book and as an encyclopedia of information on this subject the work must be considered valuable.

In any subject as controversial as backache and sciatic pain, one must expect disagreement with many things stated by any author. Perhaps for this reason Dr. Lewin has avoided any attempt to evaluate much of the information contained in the work. For one looking for means to a final diagnosis in a given case of backache the book might be disappointing. It is my feeling that in some respects the author might have been more specific along this line. Perhaps he would have subjected himself to more criticism by so doing than by following the course he has chosen.

The book is well printed and quite well illustrated with good photographs, drawings, and charts, all of which help to make clear the picture that Dr. Lewin is trying to present.

This volume is one that has been needed for a long time. While it cannot be expected that one could publish a volume on as controversial a subject as this that would be acceptable to all, it seems to me that the work is well done and will be a useful reference book to all interested in the subject of backache.

The Arthropathies. A Handbook of Roentgen Diagnosis. By Alfred A. de Lorimier, A.B., M.A., M.D., Colonel, Medical Corps, United States Commandant, The Army School of Roentgenology, Memphis, Tenn.; formerly Director, Department of Roentgenology, Army Medical School, Washington, D. C. Cloth. Pp. 319, with 678 figures. Chicago, 1943, The Year Book Publishers, Inc. $5.50.

In the field of roentgen diagnosis the atlas method of presentation has many advantages. In the past it has suffered from the inadequacy of the illustrations both in quality and quantity. In this small book this handicap has largely been overcome, and as a result a wealth of material is presented in a very small space and in a very concise manner. Both the author and the publishers are to be complimented on this achievement. The attack on the subject is unique, as the author has not been content to confine himself to joint diseases but rather has concerned himself with all diseases affecting the joints or the portions of the bones contiguous to the joints. Thus, such widely disparate lesions as congenital syphilis and bone tumors are given consideration. There are well over 600 figures, including such unusual conditions as Baker's cyst and ainhum, but a detailed study of the commoner joint lesions is not neglected. Many of the illustrations are excellent in quality. Others, unfortunately, are not well reproduced. The labeling is adequate, although the conglomeration of four to six captions into one paragraph makes the study of the figures unnecessarily complex. There is such a wealth of material in this volume that it is impossible to criticize details. Certainly every physician will find this small pocket-sized book a storehouse of information about diseases of the joints and of the bones about them.

Medical Radiographic Technique. By Technical Service Department of General Electric X-Ray Corporation under the editorial supervision of Glenn W. Files, Director. Cloth. Pp. 365. Springfield, Charles C Thomas, Publisher. $6.00.

The expansion of the field of roentgen diagnosis and the increasing need for well-trained x-ray technicians should make this new book on x-ray technique written by technicians particularly welcome. The section on x-ray physics is well presented. It should permit a well-grounded individual to attain a good understanding of the theories underlying x-ray technique and of the construction and utilization of the various pieces of equipment necessary for x-ray diagnosis. The description of skeletal anatomy is well selected for teaching x-ray technicians. Unfortunately the same cannot be said for the section on visceral anatomy, which is too brief and rudimentary. The photographic representations of the standard positions used for different parts of the body are excellent, but too many important procedures are omitted. It is difficult to understand why a recent text on roentgen technique would omit completely such important items as bronchography, roentgenkymography, the structure and uses of the cassette, radiography of the stomach upright, as well as other less obvious matters. The illustrations are good; especially are the photographs of the various standard positions well done. The book will be useful to anyone using x-rays for examination, but is not sufficiently complete nor well enough written to displace any of the present standard texts on this subject.

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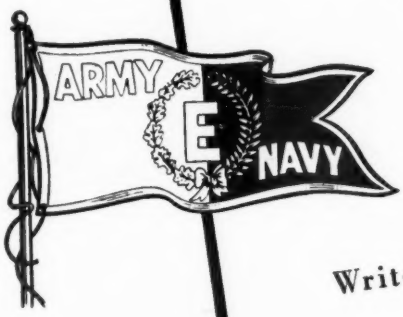

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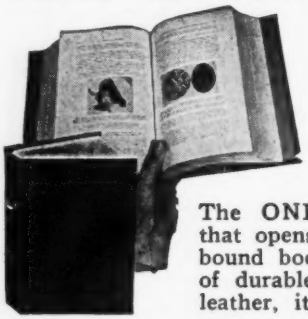

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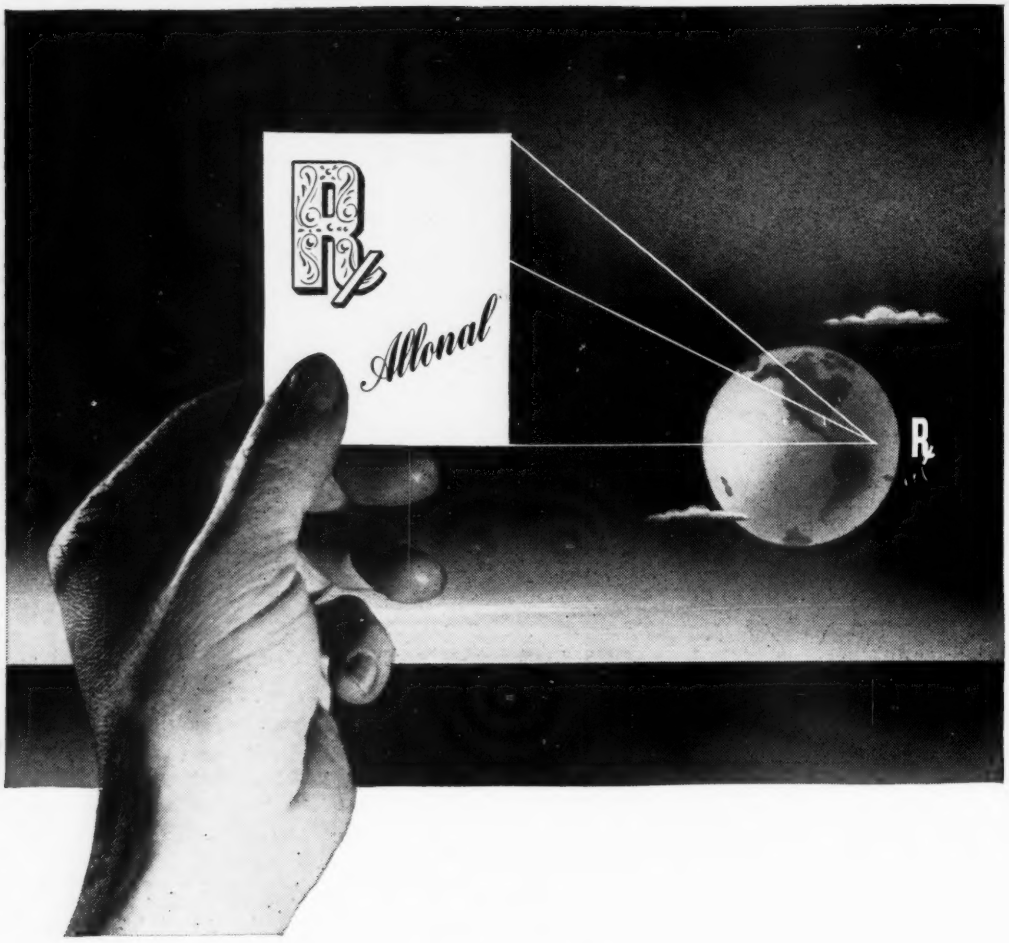

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